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AMERICAN CONTRIBUTIONS TO NOSOGRAPHY*

DAVID RIESMAN, M.D.†

PHILADELPHIA

ONE HUNDRED years ago a number of American physicians, chiefly from Boston and Philadelphia, with a few from Charleston, South Carolina, went to Paris in order to sit at the feet of the most brilliant group of medical teachers ever gathered together in one place and at one time. Laennec, Broussais, Bayle, Chomel, Andral, Bretonneau, Rayer, Alibert and Piorry were creating a new era in medicine. The humoral doctrines, the futile speculations on the nature of disease that held sway in Paris toward the close of the eighteenth century, and which were destined to dominate the German school far into the next generation, were giving place to a clinicopathological concept of disease based on a careful correlation of the findings at the bedside and the lesions observed in the mortuary. It was this new approach that appealed to the ambitious young men in this country and induced them to go to Paris in what might be called the Golden Age of French medicine. William Osler[1] in his charming essay on the influence of Louis on American medicine, and Walter Steiner,[2] of Hartford, in his recent address before the American Society of Medical History, have given interesting portraits of these Americans, the most attractive of whom, it seems, was a brilliant young Bostonian, James Jackson, Jr., Louis's favorite pupil. Fate did not permit him to fulfill the promise of his youth;‡ nevertheless he has left an imprint upon American medicine, directly through his own studies on typhoid fever, and indirectly through his father's touching memorial.[3]

Other members of the Boston group were Henry I. Bowditch, Oliver Wendell Holmes, John D. Fisher, John C. Warren, J. Mason Warren and George Cheyne Shattuck, the younger.

Among the Philadelphians were Alfred Stillé, the only one who was living when I began the study of medicine, Caspar Wistar Pennock and William Wood Gerhard, the last probably contributing more than anyone else to the spread of the French doctrines in America.§

It is reasonable to suppose that during their stay in Paris strong ties of friendship sprang up between the men from Boston and the men from Philadelphia. Indeed, we know that there was a fervent attachment between Shattuck and Stillé and between Jackson and Gerhard.‖ These friendships between the men of the two cities have descended without a break through the aftercoming generations, and I fain would believe that my presence here tonight, delivering the lecture commemorating the father of one of those Bostonians, is an expression of that century-old tradition.

The subject I have chosen, which concerns American contributions to nosography, or to the description of diseases, can be approached in either of two ways, from the point of view of original contributions to medicine, that is, of descriptions of new diseases either previously unknown or not fully differentiated, or from the point of view of the publication of texts either

*The Shattuck Lecture, delivered at the annual meeting of the Massachusetts Medical Society, Boston, May 31, 1938.

†Professor of the history of medicine, University of Pennsylvania; professor of clinical medicine, Graduate School, University of Pennsylvania; physician to Philadelphia General Hospital.

‡Some writers say that Jackson died of typhoid fever; he apparently died of dysentery.

§It is interesting to speculate why the influence of the French school, so potent in the first fifty years of the last century, began to wane, and why it was displaced by the Austro-German school. France suffered just as England from too great centralization of intellectual life in one city, the capital. Germany, with its twenty-six more or less autonomous provinces — until the World War — and with nearly as many universities, suffered politically from the absence of centralization but gained immeasurably on the cultural level. Berlin, unlike Paris, was never the only center of medicine or other branches of learning. In Leipzig, Göttingen, Heidelberg, Tübingen, Strassburg, Freiburg, Breslau, Munich — in all of them and in others the American student found scientists and humanists of the first rank. The natural rivalry prevailing among these separate intellectual groups had a profoundly stimulating effect on German culture, and in no field was this effect greater than in medicine.

Since the war, however, Minerva has found a more habitable home in this country than in Europe. Medicine is rapidly advancing here, both as science and as art. Our laboratories vie with the best in Europe and the product of research is of a high order. It is no wonder therefore that the current of wandering students now flows from East to West, and not from West to East as in colonial days and thereafter until a score of years ago.

‖Gerhard wrote home as follows about Jackson: "He has superior talents and his excellent education, conducted by his father, unquestionably the first physician in America, has cultivated his mind and developed an ardent attachment to medicine."

on a single disease or on a comprehensive subject, that became standard works in their time. I shall limit myself today, although not wholly, to the first, which in itself has grown to be a much heavier task than I had expected. I had no idea that there were so many diseases that were first described by Americans. It is a roster of which American medicine may well be proud.

I was much amazed to find on the title page of Chapman's journal (*Philadelphia Journal of the Medical and Physical Sciences*) for a period of years the following quotation from the *Edinburgh Review:* "In the four quarters of the globe, who reads an American book? or goes to an American play? or looks at an American picture or statue? What does the world yet owe to American physicians or surgeons?" Evidently the condescension of foreigners that so irked James Russell Lowell was not limited to literature. If the "Scotch Reviewer" who uttered the bitter criticism were to come back today, how amazed he would be at America's position in the world.

It is, however, only fair to say that prior to the American Revolution there was practically no medical publication of note in this country. After all, a pioneer people engaged in clearing the land and fighting the Indians had no time for creative writing. The Declaration of Independence, the United States Constitution and Washington's "Farewell Address" are sufficient credit to the young country.

The value of the few publications on medical subjects before the Revolution is purely historical and not intrinsic. Thacher's *Brief Rule* (Boston, 1677), a broadside, although it deals with medicine, was written by a clergyman. It is the only medical publication of the American colonies in the seventeenth century. "At the commencement of the Revolutionary War," says Billings,[1] "we had only one medical book by an American author, three reprints and about twenty pamphlets." The book was John Jones's *Plain, Practical, and Concise Remarks on the Treatment of Wounds and Fractures* (New York, 1776), a compilation from many sources, containing an interesting chapter on military hospitals. Somewhat more significant is Thomas Cadwallader's famous essay, *The West India Dry Gripes* (Philadelphia, 1745), printed by Benjamin Franklin. This was not a new disease — it was lead poisoning from drinking Jamaica rum made in leaden containers. The same disease affected some citizens of Boston, as Reginald Fitz[2] has shown in a charming playlet.

The first literary contributions by physicians after the close of the Revolutionary War are to be found in the *Memoirs of the American Academy of Arts and Sciences* (Boston, 1785) and in the *Transactions of the American Philosophical Society* (Philadelphia, 1786). As a strictly medical contribution the honor of priority belongs to *Cases and Observations by the Medical Society of New Haven County in the State of Connecticut* (New Haven, 1788). This is a collection of twenty-six articles by physicians, of which one is of outstanding historic importance, for it represents the first original contribution to nosography by an American physician. I refer to an article* on congenital hypertrophic stenosis of the pylorus by Hezekiah Beardsley, a native of Stratford, Connecticut, who was both druggist and physician, and practiced medicine in Southington, Hartford and New Haven. He was an original member of the Medical Society of New Haven County, founded in 1784.†

The honor of discovering this disease has been claimed for other men; for example, for George Armstrong, who in a book entitled *An Essay on Diseases Most Fatal to Infants* (London, 1771), describes the case of a child about three weeks old who died of the "watery gripes," and in whose stomach Armstrong found the pylorus to be firm. The intestines were remarkably empty. It is probable that this was a case of congenital hypertrophic stenosis of the pylorus, but Armstrong's description does not compare with that of Beardsley.‡ I reproduce the latter's article on account of its historical importance and the simplicity of its diction.

> A child of Mr. Joel Grannis, a respectable farmer in the town of Southington, in the first week of its infancy, was attacked with a puking, or ejection of the milk, and of every other substance it received into its stomach almost instantaneously, and very little changed. The feces were in small quantity and of an ash color, which continued with little variation till its death. For these complaints a physician was consulted, who treated it as a common case arising from acidity in the prima via; the testaceous powders and other absorbents and correctors of acid acrimony were used for a long time without any apparent benefit. The child, notwithstanding it, continued to eject whatever was received into the stomach, yet seemed otherwise pretty well, and increased in stature nearly in the same proportion as is common to that state of infancy, but more lean, with a pale countenance and a loose and wrinkled skin like that of old people. This, as nearly as I can recollect at this distance of time, was

*Reprinted in the Arch. Pediat. 20:355-357, 1903.

†I have been unable to locate a picture of Beardsley.

‡Cautley and Dent (Lancet 2:1873-1875, 1902) claim that the first case of congenital hypertrophic stenosis of the pylorus was described in 1841. In this they are not correct.

Kellett, of Newcastle-on-Tyne, in an interesting historical article (Arch. Dis. Child. 8:323-328, 1933) has unearthed three cases antedating Beardsley's — that of George Armstrong, mentioned above, one by Patrick Blair (Philosoph. Tr., Roy. Soc. [London] 30:631. 1717) and one by Christopher Weber (Inaug. Thesis. Göttingen, 1758). In addition he cites a possible case recorded a hundred years earlier by Fabricius Hildanus (*Opera Omnia*, Frankfort, 1646). In the last instance the child recovered under medical treatment which leaves a doubt as to its true nature; the clinical description, however, is such that the diagnosis of stenosis of the pylorus is compatible with it.

his appearance and situation when I was first called to attend him; he was now about two years old. I was at first inclined to attribute the disorder to a deficiency of the bile and gastric juices, so necessary to digestion and chylification, joined with a morbid relaxation of the stomach, the action of which seemed wholly owing to the weight and pressure of its contents, as aliment taken in small quantities would often remain on it, till, by the addition of fresh quantities the whole, or nearly all, was ejected; but his thirst, or some other cause, most commonly occasioned his swallowing such large draughts as to cause an immediate ejection, and oftentimes before the cup was taken from his mouth. It did not appear that he was attended with nausea or sickness at his stomach, but he often complained that he was choked, and of his own accord would introduce his finger or the probang, so as to excite the heaving of the stomach and an ejection of its contents; the use of this instrument was generally necessary if the stomach did not of itself, in a few moments discharge its contents; the choking would in that space of time become almost intolerable, which by this discharge was entirely removed. In this situation, with very little variation of symptoms he continued till death closed the painful and melancholy scene, when he was about five years of age. He was uncommonly cheerful and active considering his situation. A number of the most respectable medical characters were consulted and a variety of medicines was used to little or no effect. His death, though long expected, was sudden, which I did not learn till the second day after it took place. This late period, the almost intolerable stench, and the impatience of the people who had collected for the funeral prevented so thorough an examination of the body, as might otherwise have been made. On opening the thorax, the esophagus was found greatly distended beyond its usual dimensions in such young subjects; from one end to the other of this tube, between the circular fibres which compose the middle coat, were small vesicles, some of which contained a tablespoonful of a thin, fluid like water, and seemed capable of holding much more. I next examined the stomach, which was unusually large; the coats were about the thickness of a hog's bladder when fresh and distended with air; it contained about a wine pint of a fluid exactly resembling that found in the vesicles before mentioned, and which I supposed to have been received just before his death. The pylorus was invested with a hard, compact substance, or schirrosity, which so completely obstructed the passage into the duodenum, as to admit with the greatest difficulty the finest fluid; whether this was the original disorder, or only a consequence, may perhaps be a question. In justice to myself I ought to mention, that I had pronounced a schirrosity in that part for months before the child's death. On removing the integuments of the abdomen, I was struck with the appearance of the vesica fellis, which was nearly five inches in length and more than one in diameter; it lay transversely across the abdomen, and was bedded into the small intestines, which were sphacelated wherever they came in contact with it; its contents were rather solid than fluid, and resembled flesh in a highly putrid state; its color was that of a very dark green, like the juice of the night-shade berry, and a fluid of the same color exuded through its sphacelated coats. The necessity there was of interring the body that evening, put a stop to any further examination.

I should have been happy, gentlemen, if I had been able to have given you a more particular and accurate description of this very singular case, but the above mentioned circumstances forbade.

The next original contribution is that of John C. Otto,[6] of Philadelphia, on hemophilia or bleeder's disease. This strange malady must have been known from early days, since it was no doubt encountered among Semitic peoples employing ritual circumcision, and may have been a cause of death from trivial injuries among any individuals who had the taint in their blood. But it was left

John Conrad Otto
1774-1844

for Otto to give a clear description of the disease and to point out its peculiar hereditary transmission in the female line. He was a grandson of Bodo Otto, who came to this country from Germany in 1755 and served as hospital surgeon during the Revolutionary War, afterward practicing in Reading, Pennsylvania. I reproduce the essential parts of Otto's original article.

About seventy or eighty years ago, a woman by the name of Smith settled in the vicinity of Plymouth, New Hampshire, and transmitted the following idiosyncrasy to her descendants. It is one, she observed, to which her family is unfortunately subject, and had been the source not only of great solicitude, but frequently the cause of death. If the least scratch is made on the skin of some of them, as mortal a hemorrhagy will eventually ensue as if the largest wound is inflicted. The divided parts, in some instances, have had the appearance of uniting, and have shown a kind dis-

position to heal; and, in others, cicatrization has almost been perfect, when, generally about a week from the injury, an hemorrhagy takes place from the whole surface of the wound, and continues several days.

Various remedies have been employed to restrain the hemorrhagies — the bark, astringents used topically and internally, strong styptics, opiates, and, in fact, all those means that experience has found serviceable have been tried in vain.

It is a surprising circumstance that the males only are subject to this strange affection, and that all of them are not liable to it. Some persons, who are curious, suppose they can distinguish the bleeders (for this is the name given to them) even in infancy; but as yet the characteristic marks are not ascertained sufficiently definite. Although the females are exempt, they are still capable of transmitting it to their male children, as is evidenced by its introduction, and other instances, an account of which I have received from the Hon. Judge Livermore, who was polite enough to communicate to me many particulars upon this subject. This fact is confirmed by Drs. Rogers and Porter, gentlemen of character residing in the neighbourhood, to whom I am indebted for some information upon this curious disposition. When the cases shall become more numerous, it may perhaps be found that the female sex is not entirely exempt, but, as far as my knowledge extends, there has not been an instance of their being attacked.

Dr. Rush has informed me, he has been consulted twice in the course of his practice upon this disease. The first time, by a family in York, and the second, by one in Northampton county, in this state. He likewise favoured me with the following account, which he received some years since from Mr. Boardley, of a family in Maryland, afflicted with this idiosyncrasy.

A.B. of the State of Maryland has had six children, four of whom have died of loss of blood from the most trifling scratches or bruises. A small pebble fell on the nail of a forefinger of the last of them, when at play, being a year or two old: in a short time the blood issued from the end of that finger, until he bled to death. The physicians could not stop the bleeding. Two of the brothers still living are going in the same way; they bleed greatly upon the slightest scratch, and the father looks every day for an accident that will destroy them. Their surviving sister shows not the least disposition to that threatening disorder, although scratched and wounded. The father gave me this account two days since, but I was not inquisitive enough for particulars.

A full third of a century elapses before we find in American literature the next important contribution to nosography. I have reference to the classical article by William W. Gerhard[7] in which he differentiates typhus and typhoid fever. Gerhard, after studying with Joseph Parrish in the latter's private school in Philadelphia, was graduated from the Medical School of the University of Pennsylvania.* As I have already stated, he was one of the group who went to Paris to work under the great Louis. In Paris he saw what the French called typhus fever, really typhoid fever, and afterwards in England, Scotland and Ireland he observed what was called typhus fever there. He was conscious of a difference between the two. On his return to Philadelphia he was appointed one of the physicians to the Philadelphia General Hospital and had the opportunity almost immediately of studying an outbreak of typhus fever. He had no difficulty in recognizing that the typhus fever he saw in Blockley (a popular name for the Philadelphia General Hospital) was different from the typhus fever he had seen in Paris. With painstaking thoroughness he established with finality the differences between the two diseases.

*See the excellent biography of Gerhard by W. S. Middleton (Ann. M. Hist. 7:1-18, 1935).

William Wood Gerhard
1809-1872

I pause here to dwell briefly on the interesting development of our knowledge of typhoid fever as it took place in the first third of the nineteenth century. Such a study reveals the fact, not generally known, that it was Americans — men like Gerhard and Pennock in Philadelphia, and Bowditch, Shattuck and Jackson in Boston, and Bartlett, the Rhode Island philosopher — who had the clearest appreciation of the nature of typhoid fever and of febrile diseases in general. The study further demonstrates the almost unbelievable change that nosography has undergone in a little over a century.

Sauvages[8] in his ten-volume work classifies diseases after the Linnéan system of plant classification and makes about twenty-five hundred varieties or species of diseases. Alibert[9] reduces the number of families to ten, as follows: "gastroses, entéroses, chloroses, uroses, pneumonoses, angioses, leucoses, adénoses, hetmophécoses, blennoses." The first family, the "gastroses," is divided

into thirteen genres or genera, which are again subdivided into species. Genera are designated by the following terms: "polyorexie, hetérorexie, dysorexie, polydipsie, adipsie, lientérie, antemésie, gastéralgie, gastrite, squirrogastrie, gastrobrosie, gastrocélie." The species in the first genus are: "polyrexia bovina, canina, lupina," a classification based on analogy to the appetites of cattle, dog and wolf.

Pinel,[10] in a three-volume work, also adopts a system of orders. The first is that of fevers, among which he describes inflammatory fever, biliary or gastric fever, pituitous or mucous fever, putrid or adynamic fever, and malignant or ataxic fever. As late as 1820 Percy[11] described one hundred and fifteen different kinds of fever. All these classifications — and there are several more — were made by men who speculated about disease in their libraries and, unlike Sydenham a hundred years earlier, did not investigate it at the bedside and in the anatomical theater.*

Laennec and Louis, basing their work on objective clinical and postmortem studies, inaugurated a classification of diseases much more rational, and one that served its purpose until the bacteriological era, when through the discovery of the causes of infectious diseases definition and classification became infinitely easier and simpler.

The work of Laennec, Louis and others of the Paris school, as I have intimated, was brought to this country by Bowditch, Jackson, the younger Shattuck, Gerhard, Pennock and Stillé, and was taken up by American physicians with commendable alacrity — very different from a man so distinguished as William Stokes, of Dublin, who lived until 1878 but still looked upon fever as a disease instead of as a symptom of a variety of diseases.

At this point I want to recall from oblivion an American essay on typhoid fever that impresses me as one of our medical classics. It is a little booklet by Nathan Smith[12]† on typhous (tvphoid) fever. While nothing is said about the pathology or lesions of the disease, the symptoms are described in a manner not surpassed by any other writer. He knew the spots, the dicrotic pulse and the cause and time of the hemorrhages, and makes the statement that he could go blindfolded into a room where there was a case of typhoid fever and diagnose it by the smell. His treatment was conservative. He did not favor promiscuous bleeding, gave milk and water as a diet, and used water externally to reduce fever. He also noticed that patients who were constipated during the course of typhoid fever had a much better chance for recovery than those who had diarrhea — an observation that I made many times in Philadelphia during the typhoid era. What were his results? He and Dr. Perkins had sixty cases of typhoid fever in and about Dartmouth College during a local epidemic. Of these they lost only one.

The teachings of the French school and its American disciples culminated in Elisha Bartlett's[14]‡ work on fevers, truly a masterpiece.

Bartlett, instead of Percy's one hundred and fifteen types of fever, recognized only four, namely typhus, typhoid, periodical or malarial fever, and yellow fever. He refers approvingly to the theory of the cryptogamic origin of typhoid and other fevers, agreeing with, though not mentioning, John K. Mitchell,[15] father of S. Weir Mitchell, who a few years earlier had advanced the same hypothesis as an explanation of malaria.

In 1833 there was printed a volume by William Beaumont,[16] a United States Army surgeon, which entitles its author to a place in the American Pantheon. The story of his patient work with the elusive Alexis St. Martin has often been told. If ever "chance and the prepared mind" met together at the proper moment, it was when the Canadian *habitant,* Alexis, shot in the stomach, came under the care of the Connecticut Yankee, William Beaumont. Beaumont realized the opportunity of studying human digestion under hitherto unrealized conditions, and began a work that occupied him, with interruptions, for a period of eight years. He made a total of two hundred and thirty-eight observations and experiments which broadened enormously our understanding of the functions of the stomach. Not until Pavlov appeared on the scene did our knowledge of digestion receive a comparable increment.

Although not a discoverer of new diseases, Dan-

*In reading of the practice of the Paris school I found that in the time of Chomel a controversy was going on as to the value of purgatives in the Hamilton, of Edinburgh. Put to the test in the Hôtel Dieu the results were published as follows:

> In M. Chomel's practice the mortality was 1 to 3.4871794; in that of M. Bouillaud, or rather in the small number of cases reported by him, 1 to 6.2; and in that of M. Piedagnel, 1 to 7.0526315789473684210. (The decimal runs to this great length before it begins to repeat.)

These perfectly absurd figures, evidently meant as satire, are quoted from the *Lancet* in the *Medical Magazine* (3:685-688, 1835).

†One of Nathan Smith's pupils was George Cheyne Shattuck, the founder of the Shattuck Lecturership. Gross[13] called Nathan Smith the best all-around American physician of his time.

‡Bartlett was born in Rhode Island. He studied one year in Paris and afterward held positions in seven medical schools — in Woodstock, Vermont, Pittsfield, Massachusetts, Hanover, New Hampshire, Baltimore, Lexington and Louisville, Kentucky, and New York City. He attributed the differentiation of typhoid fever and typhus chiefly to Gerhard, but had great praise for Louis, of whom he wrote as follows:

> The recognition of typhoid fever as distinct from typhus is largely due to the teachings of Louis in Paris and his accurate description of symptoms and lesions. Louis' "Researches" first translated by Bowditch constitute one of the few imperishable monuments that have from time to time, and at distant intervals, been raised up along the pathway of our science.

It was with much interest and gratification that I found the following eulogy of Nathan Smith's essay in Bartlett's book:

> To an American practitioner, it is worth infinitely more than all the modern English treatises on fever put together. Nathan Smith was a shrewd, clear-headed, patient and careful student of nature — his vision undazzled and his judgment never perverted by fanciful speculations; and the labor of my book will not be wholly lost, if it succeeds in some degree in calling back the attention of my countrymen to his neglected and almost forgotten pages.

iel Drake deserves to rank among the great men of medicine in this country who have contributed to nosography. Born and reared in poverty, he obtained the rudiments of a medical education as an apprentice. Twice he entered the University of Pennsylvania and finally got his degree of M.D. in 1815, at the age of thirty years. He was a wanderer all his life, changing his locality as a teacher no less than seven times. Two medical colleges were founded by him, the Medical College of Ohio (1819) and the Cincinnati Medical College (1834). His principal fame rests on his monumental work, *Diseases of the Interior Valley of*

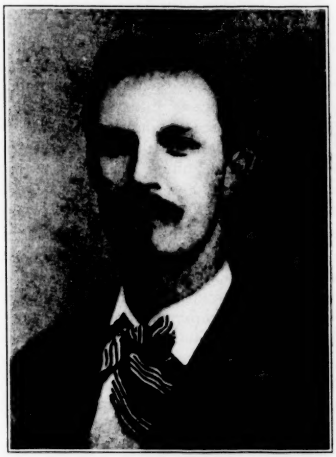
Daniel Drake
1785-1852

North America) Cincinnati, 1850; Philadelphia, 1854). As an attempt to relate geography, topography and climate to diseases it is the first book of importance since Hippocrates' *On Airs, Waters and Places.*

William Currie, of Philadelphia, a scholarly physician who had studied under John Kearsley, wrote two works entitled *Historical Account of the Climates and Diseases of the United States* (Philadelphia, 1792) and *View of the Diseases Most Prevalent in the United States* (Philadelphia, 1811). Neither these nor the compilation of Noah Webster entitled *A Brief History of Epidemic and Pestilential Diseases, with the Principal Phenomena of the Physical World* (Hartford, 1799), can in the remotest way be compared with Drake's masterpiece. Noah Webster was not a physician; he is best known as the great American lexicographer.

Another long hiatus confronts us, and then we come to the discovery, nosographically speaking, of Huntington's chorea. This devastating disease originated on Long Island. The families that brought it over, according to the recent careful studies of Vessie,[17] came from a small village, Bures, in Suffolk, England, and consisted principally of three men and their wives who landed here in 1630. From the very beginning these people, who are not definitely identified in Vessie's article, appearing under the fictitious names of Wilkie, Nichols, Jeffers and Jefferson, had a bad record, the men being vagrants or criminals, and many of the women prostitutes. A number were tried for witchcraft during the witch-burning era. From these degenerate settlers and their immediate offspring practically all the cases of adult chorea of hereditary type are descended.

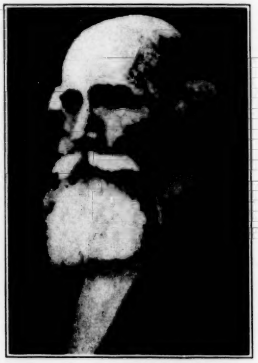
The earliest published reference to the disease, according to Russell N. deJong,[18] occurs in a letter written by Charles Oscar Waters in 1841, shortly after his graduation from Jefferson Medical College, to Robley Dunglison. Waters's brief account of the disease, which was known among the common people as the "magrums," is clear



George Sumner Huntington
1862-1927

enough to establish the fact that he had seen instances of hereditary chorea. Waters practiced for a time in Missouri and Iowa and then entered the Presbyterian ministry. Another reference is by Charles Rollins Gorman in his inaugural dissertation presented to the Faculty of Jefferson Medical College and published in the third edition of Dunglison's *Practice of Medicine* (Philadelphia, 1848). However, the description given by George Huntington, at the time practicing in Pomeroy, Ohio, is far superior to that of his predecessors, so that no canons are violated in calling it by his name.

Huntington was born in East Hempstead, Long Island, where his father and grandfather had observed the disease. After a stay in Ohio he returned East and practiced in Dutchess County, New York. In his article,[19] published in 1872, he calls attention to the three characteristic features of the malady: its hereditary nature, its tendency to lead to insanity, and the fact that it comes on as a grave disease only in adult life. The disease was spoken of by those in whose veins the seeds were known to exist with a kind of horror, and was not alluded to at all except through dire necessity, when it was mentioned as "that disorder." It is commoner among men than among women. The literature on Huntington's chorea is extensive. A valuable series of articles by James M. Winfield, Adolf Strümpell, M. Lannois, J. Paviot, William Osler, Smith E. Jelliffe, Frederick Tilney, A. R. Diefendorf and William Browning was published in *Neurographs*.[20]



John M. Riggs
1810-1885

It is not surprising that an important disease of the teeth should have been first recognized, at least carefully studied, by an American dentist. John M. Riggs was born in Seymour, Connecticut; he studied medicine at Jefferson Medical College, and dentistry with Horace Wells, of Hartford, Connecticut. Riggs pulled a tooth for Wells, who had taken nitrous-oxide gas, the first time that such an extraction was done under that anesthetic. Riggs[21] gives an exhaustive description of inflammation of the gums and absorption of the gums and of the alveolar process. He is conscious of the fact that the disease is not new, but his description is complete and has not been surpassed. That is why the disease, pyorrhea alveolaris, is also called Riggs's disease.

In the last quarter of the nineteenth century medical men in Philadelphia were probably more active than any other group in the country. This was due in part to the existence in the city of several flourishing medical schools, in part to the unique position occupied by the medical publishers of the city. That general pre-eminence does not exist any longer, due, I trust, to the fact that other cities have crept up on Philadelphia in activity and reputation and not to any slipping back of the city. This brings me to speak of the work of S. Weir Mitchell, probably the most distinguished physician of his time, whose observant eye saw a number of new diseases. In 1878 he[22] published his famous essay on the red disease of the extremities, erythromelalgia, a vasomotor neurosis.* The disease is commonest in men and affects principally the legs, the chief symptoms being heat, redness and a throbbing pain. Mitchell[23] apparently was the first to recognize eye-strain headache.

Silas Weir Mitchell
1829-1914

While not strictly nosographical, his rest-cure treatment, first announced in a small book entitled, *Wear and Tear* (Philadelphia, 1873), and published afterward as *Fat and Blood* (Philadel-

*The name of this disease was suggested to Mitchell by John Ashhurst, professor of surgery at the University of Pennsylvania, and an erudite classical scholar.

phia 1877), was an important American contribution to medicine and procured for him a world-wide fame and an enormous practice.

In association with Morehouse and Keen, Mitchell[24] made valuable observations during the Civil War on the effect of gunshot injury to nerves. He called attention to the burning pain — causalgia — so common after such injuries.

Another Philadelphia contribution, not of great importance but worthy to be mentioned, is the so-called Morton's toe. Thomas G. Morton, son of Samuel G. Morton, the first American anthropologist, was one of the surgeons of the Pennsylvania Hospital and a pioneer operator in appendicitis. He[25] described a series of cases of pain in the feet due to pressure on the nerves between the third and fourth toes. I have seen several instances of metatarsalgia or Morton's toe, and believe it to be a small but definite entity.

A contribution of signal importance was made during the Civil War by the Philadelphia clinician, Jacob M. Da Costa.[26] Da Costa was a native of St. Thomas, West Indies; he received his medical education in the Jefferson Medical College and in European universities. As professor in his Alma Mater and as physician to the Pennsylvania Hospital he became the revered teacher of thousands of students. He was a member of a great medical faculty, which included Samuel D. Gross, Joseph Pancoast and William W. Keen, three of the most famous surgeons in our history. Da Costa's classical essay to which I refer was entitled, "Observations on the Diseases of the Heart Noticed Among Soldiers, Particularly the Organic Diseases." In this essay he devoted a section to what he called the "irritable heart," which he later expanded in his textbook on medical diagnosis. An excerpt from the latter is as follows:

> Those who are subject to the disorder complain of headache, dizziness and palpitation. The last comes on chiefly during exercise but may also take place when the patient is quiet. The pain is generally dull and constant and is often also described as shooting and as taking place only in paroxysms. The action of the heart is very rapid and in many instances its rhythm is irregular. The impulse is slightly extended but not forcible, like that of hypertrophy; it is rather abrupt and jerky. The sounds are modified as follows: the first sound is short, sometimes sharp, resembling the second sound; at other times it is extremely deficient and hardly recognizable; the distinctness of the second sound is much heightened. We hear either no murmurs in the heart or in the neck, or they are inconstant. The pulse is much influenced by position, falling rapidly, twenty beats or more, when the erect posture is exchanged for the recumbent. The disorder is very obstinate and improvement comes but slowly. The cause of the morbid cardiac impressibility is difficult to ascertain. It seems in many instances to have followed fatiguing marches; in some, to have occurred after fevers or diarrhea; it was not connected with scurvy, or with the abuse of tobacco. That it was not due to anemia was proved by the general aspect of the men, which was often that of ruddy health. Similar conditions of the heart may result from excessive dancing, excessive smoking, and certain occupations, such as glass-blowing.

The condition seemed to be more or less forgotten until the World War, when it became an important factor in the invalidism of men in the service. It was then called by the high-sounding name of "neurocirculatory asthenia" in the American Army and "disordered action of the heart" in the British Army; but none of the modern descriptions excel that of Da Costa.

Da Costa was the author of a popular textbook on physical diagnosis, which passed through nine

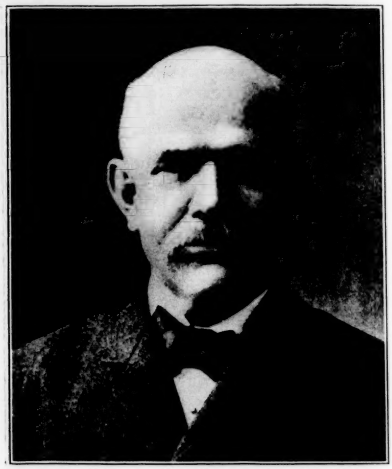
George Miller Beard
1840-1883

editions. As a consultant Da Costa was most punctilious in observing the traditional formalities. I recall a consultation in which there were seven physicians — as the patient was the wife of a doctor the number is readily accounted for — I happened to be the sixth in sequence. Da Costa insisted that we enter the sickroom exactly in the order of summons; he came last — a definite Philadelphia custom. We left the room in reverse order.

Neurasthenia, or nervous exhaustion, was raised to the dignity of a clinical entity by an American whose name is almost forgotten at the present time. George M. Beard[27] as early as 1869 published a paper on this affection. He referred to it again in his and Rockwell's work *Electricity*, but the subject excited no interest in the profession in this country. In England and in Germany, on the other hand, Beard's writings met with criticism and comment, at once sympathetic,

intelligent and scientific. Beard later published a substantial volume entitled *A Practical Treatise on Nervous Exhaustion* (New York, 1880). In this work he stated that nervous exhaustion (neurasthenia) was commoner in this country than any other form of nervous disease, but it was only vaguely recognized and referred to as general debility, nervous prostration, nervous debility, cerebral and spinal anemia, and hyperemia.

At the present time the concept of neurasthenia is still vague, although all textbooks accept it as a clinical entity. However, as our knowledge increases, neurasthenia as a disease entity may join hypochondriasis, ptomaine poisoning and others in the limbo of deceased diseases. As understood, it may be defined as a condition of pathologic weakness without a discoverable organic lesion and characterized by undue fatigability and a tendency to mental depression, as well as an undue instability to ordinary stimulation. Few human beings go through life without at some time experiencing the disease so well described by Beard.



Reginald Heber Fitz
1843-1913

The history of appendicitis is old. It goes back to a case observed by Heister in 1711.* The first case of perforation of the appendix was recorded in 1759 by Mestivier.[28] The slowness with which so important a condition became appreciated was due to the fact that men were afraid to open the abdominal cavity. As Georg Wegner,[29] a Berlin surgeon, said early in the nineteenth century, "My companions as well as myself have all been brought up in the fear of the Lord and of the peritoneum."

In June, 1886, Reginald H. Fitz, of Boston, read an essay at the inaugural meeting of the Association of American Physicians entitled "Inflammation of the Vermiform Appendix."[30] This masterly essay focused the attention of medical men upon appendicitis. It is an index of Fitz's thoroughness that he makes fifty-eight citations from the literature in this article. A. L. Loomis at the same meeting called the essay a "positive paper." In closing the discussion Fitz uttered a statement that sounds strange to modern ears: "So impressed have I been with the obscurity of the signs of incipient inflammation of the appendix that no attempt has been made to consider the process until there was evidence that perforation had taken place." But it was not long until through the work of Charles McBurney, John B. Murphy, John B. Deaver, Maurice H. Richardson and others, America led the world in the diagnosis and treatment of appendicitis. Unfortunately, for reasons I cannot go into here, there has been a disconcerting increase in mortality from the disease in recent years.

An amusing contretemps occurred between Thomas G. Morton, of Philadelphia, and Sir Frederick Treves, of London, as to who deserved the credit for priority in operating for appendicitis. Morton[31] claimed that he was the first to operate purposively and that the procedure should be called Morton's operation. Sir Frederick[32] with gentle irony replied that his tardiness in making known his own operation, performed a few months earlier, should be excused seeing that he lived "in a remote island [Great Britain]."† Neither, however, is entitled to credit for priority, if such a thing were important, for in July, 1883, F. A. Mahomed,[33] of London, advised operation in a case of appendicitis and worked out the steps of the operation, which was performed by Charters J. Symonds.

Acute pancreatitis is another disease with which Fitz's name will be forever connected. He was not its discoverer, but he brought together the scattered literature, and with that and his own experience as a basis wrote an essay[34] that has become a classic. Not only are the symptoms clearly outlined but the morbid anatomy, including the fat necrosis,

*The appendix itself was overlooked by the early anatomists, but Lambert (Ann. Med. Hist. 9:422-427, 1937) has recently pointed out that Vesalius not only pictured it but actually described it in his *Fabrica*, although many have said that he did not do so. Vesalius called it the blind intestine, caecum intestinum.

†Treves operated purposively on February 16, 1887, the case being reported February 14, 1888. Morton's first operation was on April 25, 1887. It is not stated in Morton's report that a correct diagnosis was made before operation, though that claim was later advanced.

is fully described. The article concludes with a brief recital of a case in which Fitz made the correct diagnosis, confirmed at autopsy. The majority of the consultants had agreed on a diagnosis of acute intestinal obstruction.

So far nearly all the original observations of diseases have come from the Atlantic seaboard, but the West was not inactive. In 1892 W. F. Milroy,[35] professor of clinical medicine and hygiene in the Omaha Medical College in Omaha, Nebraska, published an article that has enshrined his name forever in the history of medicine, since by that article he became the father of Milroy's disease. During the course of an insurance examination of an American clergyman, thirty-one years of age, who had never been sick, Milroy found as the only abnormality edema involving the feet and extending up the legs to the knees. The applicant stated that this enlargement was a family characteristic, which had been inherited from his mother. Fortunately for the purposes of Milroy's study, the mother's family had been long in America. In 1883 a member of the family published a volume giving the family history in America for two hundred and fifty years. The abnormality described seems to have entered the family by marriage about 1768. With the aid of the volume and the assistance of the relatives then living, Dr. Milroy was able to make a study of six generations, comprising ninety-seven individuals, of whom twenty-two, or about twenty-three per cent had swelling of the legs. Of these twelve were men and seven women, the sex of three cases being unknown. The invariable characteristics of the disorder were as follows:

Congenital origin, with a steady growth corresponding to the normal growth of the body until adult size was attained.

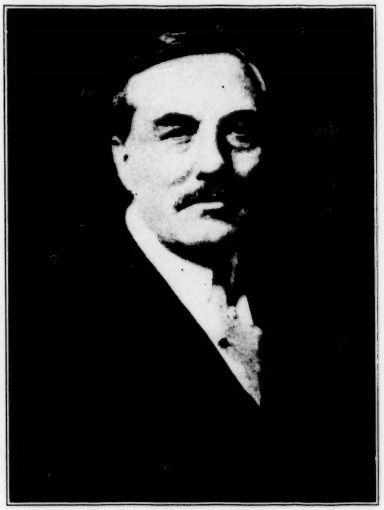
The limitation of the edema to one or both lower extremities, the area involved varying.

Permanence of the edema.

Entire absence of constitutional symptoms or local symptoms, aside from those described.

At the time of the publication of Milroy's paper, a great deal of interest had been created in angioneurotic edema through a paper by William Osler,[36] published in 1888. In Milroy's judgment, while the condition corresponded in a considerable degree to angioneurotic edema, it was distinguished from it by the four characteristics just cited.

Frank Billings, of Chicago, was for his time and region what Daniel Drake had been in his life for the Ohio Valley. Billings's principal contributions to American medicine were the improvement of medical education and the concept of focal infection. The latter is one of the most important American contributions to medicine in our time. In his first article Billings[37] enumerates the sites of focal infection as follows: the tonsils, the teeth, the sinuses in the head, bronchiectatic and pulmonic cavities, the gastrointestinal tract (chronic ulcers), the appendix, the gall bladder and ducts, the urinary tract, more particularly the prostate gland, the genital tract, and, finally, anywhere in the body. The systemic results of such focal infection are chronic arthritis, nephritis, cardiovascular degeneration, chronic neuritis and myalgia. Billings insisted that focal infection should always



Frank Billings
1854-1932

be looked for, and when it seemed related to the systemic disease, radical measures should be instituted to remove it.

Very little has been added to the clinical aspects of the subject since Billings's early writings. Through the work of Edward C. Rosenow and other bacteriologists working in co-operation with Billings, the principle of focal infection has been put on a secure foundation.

Billings's ideas, strangely enough, have not taken the hold in Europe that they have in this country, although more attention is given to them by European physicians now than was the case in former years. Nevertheless, these men still ridicule what they consider our passion for excising tonsils and extracting teeth.

To the concept of focal infection — that effects

at a distance are produced by toxins or by bacteria from the original focus — the concept of allergy has been added. It is now held that through the existence of a local focus an allergic state, a condition of sensitization to toxins, may be established.

I cannot leave this subject without giving an illustration of the universal fact that there is nothing new under the sun. When one goes back in medical literature, one often finds that what seems new

Benjamin Rush
1745-1813

has deep roots in the past. Billings himself admitted that there was nothing new in the principle involved in his concept.

It is interesting that the germ of his theory may be found in the works of Benjamin Rush.[38] Rush writes as follows:

> Some time in the month of October, 1801, I attended Miss A. C. with a rheumatism in her hip joint which yielded, for a while, to the several remedies for that disease. In the month of November it returned with great violence, accompanied with a severe toothache. Suspecting that the rheumatic affection was excited by the pain in her tooth, which was decayed, I directed it to be extracted. The rheumatism immediately left her hip, and she recovered in a few days. She has continued ever since to be free from it. . . .
>
> When we consider how often the teeth, when decayed, are exposed to irritation from hot and cold drinks and aliments, from pressure by mastication and from the cold air, and how intimate the connection of the mouth is with the whole system, I am disposed to believe they are often the unsuspected causes of general, and particularly of nervous, diseases. When we add to the list of those diseases the morbid effects of the acrid and putrid matters which are sometimes discharged from carious teeth, or from ulcers in the gums created by them, also the influence which both have in preventing perfect mastication, and the connection of that animal function with good health, I cannot help thinking that our success in the treatment of all chronic diseases would be very much promoted by directing our inquiries into the state of the teeth in sick people, and by advising their extraction in every case in which they are decayed. It is not necessary that they should be attended with pain in order to produce disease, for splinters, tumours and other irritants before mentioned often bring on disease and death, when they give no pain and are unsuspected as causes of them. This translation of sensation and motion to parts remote from the place where impressions are made appears in many instances, and seems to depend upon an original law of the animal economy.

Considering that this was written one hundred and thirty-five years ago, it entitles Rush, who has lately suffered at the hands of biographers, to our unqualified respect and admiration.

But even the work of Rush is not the first that I was able to find. The French surgeon, J. L. Petit, to whom we owe the terms "pericranium" and "periosteum," in his *Traité des Maladies Chirurgicales et des Operations* (Paris, 1774), published posthumously, wrote:

> Caries of a tooth is often the cause not only of small abscesses, but even of an infinity of others on the outside of the mouth, and even a variety of other diseases which appear to have little to do with the teeth.

His most interesting case is that of a young maid in the service of the Princess of Condé who had migraine that lasted five years. She was treated by many physicians at a time, as Petit says, "when bleeding was the fashion." She had at least twenty venesections, as many from the arm as from the foot, and then one was ordered from the throat. The Princess, who had been conten. to have the bleedings done by Petit's pupils, begged him to make the throat venesection himself. As he had never seen the patient before, he asked her a number of questions, and found on examining her mouth that in each jaw she had, instead of sixteen, eighteen teeth, the arrangement of which was quite irregular. He thought that the second molar on each side interfered with the others. He extracted these teeth, and to the astonishment of the Princess and of the whole world, within twenty-four hours the young woman was cured of her sickness, which had lasted five or six years.

The name of William Osler — a Canadian by birth — is so enshrined in the history of American medicine that it does not detract from his fame to say that he did not discover any important disease. He had, however, the more valuable gift of seizing the right moment to publish his personal

observations on diseases recently described, usually in a less perfect way, by others. Thus his name is attached to polycythemia vera, the first description of which was given by Vaquez. Osler himself refused to take credit for the discovery of this disease although his comment on it brought it to the attention of the medical world. Other classic articles on established diseases are those on cerebral palsies in children, on endocarditis and on angina pectoris. There is, however, one condition which Osler[39] was the first to observe, that might be called Osler's disease; yet it is not a clearly defined entity. I refer to what he called the visceral manifestations of erythema exudativum. Erythema exudativum or nodosum had long been known, but it was Osler who called attention to the visceral complications of the disease and to its marked tendency to recur. The visceral complications are usually gastric crises,—colic, with vomiting and diarrhea,—and at times acute nephritis and hemorrhages into the kidney and bowel, or even into the brain. The skin lesions are erythema multiforme, urticaria and purpura. The third feature in the Osler triad is arthritis, a swelling of the joints, especially periarticular swelling. Osler leaves the relation of the condition to rheumatism undecided. However, I have seen the syndrome in connection with mitral stenosis of rheumatic origin.

Sir William Osler
1849-1919

Osler's *Textbook of Medicine* when it first appeared in 1892 was like a breath of fresh air in a stagnant room. It was conceived by a man in his prime who, unlike any medical textbook writer gone before, was profoundly versed in two indispensable elements: knowledge of pathology and wisdom gained through a large experience with patients. And medicine has not changed in this regard. The really great teacher in the medical school or in the sickroom endeavoring to help the troubled family doctor is the man who knows pathology as well as clinical medicine.

Today, few textbooks of medicine have any striking individuality, in fact the day of the one-man textbook like Osler's or Strümpell's has gone, probably forever. The man equipped in every branch of medicine as he existed in the nineteenth century and earlier is no longer a possibility. The field has grown too vast to be tilled by any one man. Books are either written by specialists or are compiled under the supervision of an editor. Many works of both kinds exist, and meet the needs of the practicing physician.

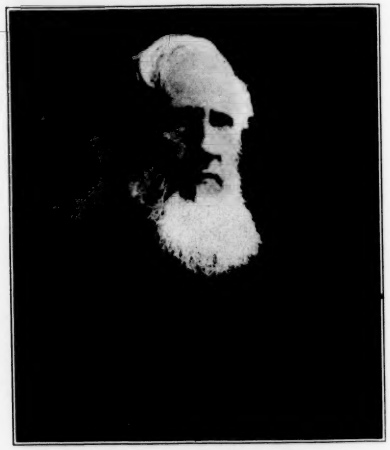
I cannot leave the subject of Osler without saying a word about his extraordinary personality and his significance for American, indeed for English, medicine; I use the adjective in the linguistic sense. It will be readily agreed that no man in modern times has exerted an equally potent influence on the medical men of his generation. In many respects Osler may be compared with Erasmus. Both were humanists; both consciously and unconsciously were teachers and inspirers of their contemporaries. Wherever Erasmus went he was received with acclaim and even adulation. Osler, likewise, met with an enthusiastic welcome on his visits to American, Canadian and English cities. Erasmus liked neither the violence of Luther nor the bitter counterattacks of Luther's antagonists. Osler also stood for tolerance; yet were he living today he would oppose with all the power of his word and pen the soul-binding ideology of totalitarianism.

Osler's influence as a medical teacher was personal and will probably wane with the passing of his pupils, but his influence as a humanist is sure to grow greater with time. He has bequeathed to us and to future generations an interest in the history of medicine and a love of books. By following him we too may become Erasmians.

In no field have the American contributions to nosography been more abundant than in that of dermatology. I ascribe this to two causes: first, the generally high order of ability of American dermatologists; second, the ease with which a disease on the surface of the skin can be studied

and accounts of it compared. Another fact which struck me in my reading of the dermatological literature was the penchant of dermatologists for giving high-sounding names to the diseases they discover. Such words as measles, mumps, chickenpox, scarlet fever and colitis would never satisfy a specialist in skin diseases. That is why one meets such names as acanthosis nigricans, acarodermatitis urticarioides, dermatitis herpetiformis, hydroa herpetiformis and herpes gestationis. They recall the time already referred to when men classified diseases as Linné classified plants; into families, orders, genera and binomial species.

One of the first original publications on a newly



Louis Adolphus Duhring
1845-1913

discovered disease of the skin was made by T. C. Gilchrist[40] in 1896. The disease is blastomycosis. The patient described had been under the care of Louis A. Duhring, of Philadelphia, who sent a specimen of tissue to Gilchrist for study. The latter found that the affection was due to a fungus, a blastomyces. His observation was soon confirmed by other dermatologists in this country and abroad. The fungus gains entrance at the site of a wound. While usually limited to the skin, the disease may become systemic, in which case it is nearly always fatal.

Duhring, a pioneer American dermatologist, was professor of diseases of the skin in the University of Pennsylvania. He was an eccentric individual, who always wore gray gloves — it is believed because of a fear of germ contamination. Little is known of his private life — he was a bachelor; after his death it was discovered that he had been a passionate speculator in the stock market, entering and leaving his broker's offices by a rear door. He must have been exceptionally successful, for he left a fortune of over a million dollars, part of it to the University of Pennsylvania for research on diseases of the skin, part to the College of Physicians of Philadelphia for its library.

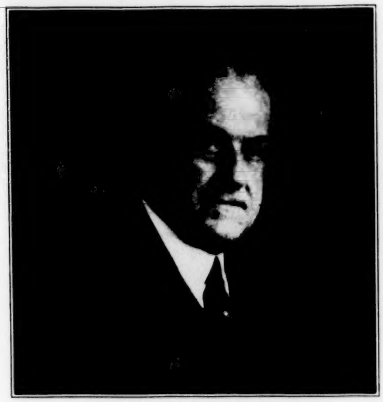
Duhring's memory is perpetuated in the disease dermatitis herpetiformis, or Duhring's disease.[41] The condition is characterized by pleomorphic lesions, vesicles, pustules and blebs, has a marked tendency to recur, and runs an altogether irregular, capricious course.

Jay Frank Schamberg
1870-1934

Duhring had many pupils. One of the most distinguished was the late Jay F. Schamberg, to whom we owe the description of two diseases, progressive pigmentary dermatosis,[42] a hereditary disease of unknown cause, and grain itch.[43] The former is a chronic disease beginning as reddish points or dots of pinhead size, forming irregular patches which extend slowly by the formation of new lesions at the periphery. The points eventually disappear, leaving a brownish or reddish-brown pigmentation, which slowly fades. There are no subjective symptoms. The cause is unknown; the disease seems to be hereditary. So far it has been found only in males. It is usually limited to the lower third of the legs and the dorsums of the feet. Grain itch, straw itch or mattress itch attacks persons sleeping on straw infested with a minute insect *Pediculoides ventricosus*. Schamberg called the disease acaroderma-

titis urticarioides parasitica. It is characterized by various types of eruption, largely urticarial, with chills, mild fever, malaise, moderate enlargement of the lymph nodes and intolerable itching.

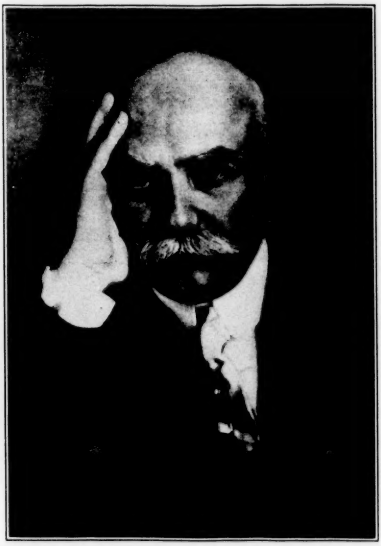
John A. Fordyce,[44] of New York, gave his name to a peculiar affection of the mucous membrane of the lips and oral cavity characterized by the presence of minute, yellowish bodies which impart to the skin of the lips and mouth a strange appearance. Fordyce, in conjunction with George H. Fox,[45] first described another disease of the skin, affecting the axillary regions and characterized by a papular eruption and intense itching. It involves chiefly the orifices of the sweat ducts, and is histologically related to porokeratosis.



John Addison Fordyce
1858-1925

Under the term acanthosis nigricans Dr. Sigmund Pollitzer,[46] of New York, described a disease of the skin of which he is considered the discoverer. He was working at the time in the famous clinic of Unna in Hamburg. The disease, which somewhat resembles ichthyosis, produces keratosis and a blackish discoloration of the skin. When it occurs in adults it is generally associated with cancer of some internal organ. There may be a relation between acanthosis nigricans and Addison's disease. Pollitzer[47] also described for the first time a recurrent eczematoid affection of the hands characterized by the occurrence of groups of resistant vesicles on the backs of the hands and the backs of the lower portions of the forearms. They dry slowly, desiccate and disappear, to be followed after varying intervals by repeated recurrences. During the eruption there is considerable itching and a moderate eosinophilia.

John T. Bowen,[48] of Boston, called attention for the first time to a disease called precancerous dermatosis, which belongs to the same class as Paget's disease of the nipple. Bowen's two cases were in men in the fifth decade. The lesions were limited in one to the left gluteal region, and in the other to the left calf. Arsenic was suspected as a possible etiologic factor.



Francis Xavier Dercum
1856-1931

I have spoken of two important hereditary diseases, hemophilia and Huntington's chorea. Another in this group is the mysterious condition called amaurotic family idiocy, the identification of which is due to Bernard Sachs,[49] of New York. The eye changes had been observed previously in 1881 by Waren Tay,[50] of London. So far the disease has been observed almost exclusively in Jewish children. The symptoms are mental impairment during the first months of life, leading to absolute idiocy, paresis or paralysis of the greater part of the body, a diminution of vision ending in total blindness, and a fatal termination before the age of two; most significant of all is the occurrence of the affection in several members of the same family.

Francis X. Dercum,[51] of Philadelphia, at the time instructor in nervous diseases at the University of Pennsylvania School of Medicine, published in 1888 an article in which he described a disease now called Dercum's disease or adiposis dolorosa. The patient in whom the affection was discovered was a native of Ireland, fifty years old, well known to the physicians attached to the Philadelphia General Hospital in the late eighties and early nineties. I saw her frequently. There was a marked enlargement of the shoulders, arms, back and sides

Christian Archibald Herter
1865-1910

of the chest through the deposit of fibrofatty masses, which hung in festoons from the arms. In some areas the flesh felt finely lobulated; in others, especially on the inside of the arms, as though it were filled with bundles of worms. The diseased tissues were sensitive to pressure. Appended to Dercum's paper is a careful microscopic study, made with the methods then available, of a little tissue obtained from both arms by trocar. Dercum thought there was some resemblance between this disease and myxedema, although the two were not identical.

Achylia gastrica is scarcely a disease; it is rather a symptom, but it has become a quasi-entity. We owe its original description to Max Einhorn,[52] of New York City. It is a condition in which there is a permanent absence of gastric secretion. At the present day it is customary to inject histamine, which will sometimes cause the secretion of hydrochloric acid when by the ordinary test meal it is absent. Although most characteristic of pernicious anemia, it may occur in other conditions. Sometimes it is familial. It has been observed in association with combined sclerosis of the spinal cord without pernicious anemia, and in rare instances it is found in cancer of the stomach.

Christian A. Herter, a native of Glenville, Connecticut, and a man of high scientific integrity, pointed out a clinical condition called intestinal infantilism. In a book[53] published in 1908 he gives the essential features as follows:

> (1) An arrest in the development of the body; (2) the maintenance of mental powers and fair development of the brain; (3) marked abdominal distension; (4) moderate grade of anemia; (5) rapid onset of physical and mental fatigue; (6) various obtrusive irregularities referable to the intestinal tract.

The result of intestinal infantilism is retardation of growth. Many children die from acute infections; others live to be pronounced dwarfs. Fortunately, such dwarfism is not incompatible with a high degree of mental development. At the time when Herter wrote, treatment had not reached a very satisfactory state. He advised a low-fat diet and stressed the value of gelatin, which today, however, is not used a great deal in infants' diets. The question arises whether in intestinal infantilism we are not dealing with a condition of vitamin deficiency, which today is treated more efficiently and more promptly. On the whole, the disease is now rare on account of a better understanding of the dietary needs of the child.

Herter was only forty-five when he died. He had a great deal to do with the planning and development of the Rockefeller Institute for Medical Research, and acted as adviser to the Department of Agriculture in Washington in the enforcement of the Food and Drug Act.

In the last few years great advances have been made in our knowledge of the physiology and pathology of the peripheral vascular system. Besides such long-known conditions as thrombosis and embolism, Raynaud's disease and erythromelalgia, there have been added Buerger's disease and acrocyanosis. More than forty years ago, Solomon Solis-Cohen,[54] of Philadelphia, under the term, vasomotor ataxia, described a condition of instability of the mechanism of the circulation, affecting both the heart and the vessels of the extremities. He concluded from analogy that a similar instability might affect the vessels of the glands and

of the viscera. Local syncope, local asphyxia, dermographism, mottling of the skin and nails, petechial spots, nosebleed, hemoptysis, hematuria and other hemorrhages were signs of Solis-Cohen's vasomotor ataxia. While the disease cannot be called a definite entity, it may be said that by his articles on the subject Solis-Cohen inaugurated the modern era of peripheral vascular diseases.

Thromboangiitis obliterans, or Buerger's disease, was discovered in 1908.[55] Buerger's disease is most frequent in Jews from Russia and Poland, although, according to the statistics of the Mayo Clinic, it appears to be increasing in the non-Jewish native population. Most of the patients smoke cigarettes to excess. Buerger's disease is an inflammatory vascular affection involving the arteries and veins, especially of the feet and legs, and leading to presenile, spontaneous gangrene. It is one of the most painful of all chronic diseases. Formerly the only treatment was amputation, but of late, methods have been discovered by which the disease can be treated successfully by more conservative measures.

Thromboangiitis obliterans is a representative of the diseases of the peripheral vascular system which in the course of a few years have achieved the dignity of a special medical discipline. Through the work of August Krogh, Sir Thomas Lewis and a group of American investigators, "angiology," as the specialty might be called, has made great strides and has its own department in many hospitals. The American contributions are of outstanding importance.

An important original American contribution is sickle-cell anemia, also called sicklemia or drepanocytic anemia. This is a strange hemolytic anemia, confined almost wholly to Negroes, in which the red blood cells under certain conditions show a peculiar sickle shape. It was first described by James B. Herrick,[56] of Chicago. Dr. Herrick, who was recently president of the Congress of American Physicians and Surgeons, is well known for his important contributions to our knowledge of coronary occlusion. He is a fine scholar, and represents the ideal combination of humanist and scientist.

Sickling of cells is found in from 5 to 7 per cent of Negroes, but in only a fraction of these does it present itself as a serious clinical condition. Lately it has been observed in a small number of pure Whites. It is hereditary and familial and can be transmitted by either sex. Sicklemia may remain latent for a long time, but when active it is characterized by recurrent phases of fever, anemia, weakness, dizziness, large spleen, a tendency to ulceration of the legs, slight jaundice, pains in the joints and muscles, and abdominal pains. The last sometimes resemble tabetic crises. The sickling is best demonstrated in a sealed preparation of fresh blood. During the active phase there is a marked anemia and a moderate leukocytosis.

Hand-Schueller-Christian's disease was discovered when in 1903 Alfred Hand,[57] of Philadelphia, at that time only one year out of medical school, reported the case of an undersized boy three years old with exophthalmus and polyuria who died of bronchopneumonia. At the autopsy peculiar yellowish spots were found in the skull cap, as well as various other lesions, including enlargement of the liver and the spleen, the former containing minute gray nodules. Subsequently Henry A. Christian,[58] of Boston, reported a case very similar to Hand's. This was also in a child, whose skull when studied by x-ray showed extensive defects in the bones. Similar but less marked changes were found in the ilium and ischium. Christian was able to collect two other cases in his search, both reported by Schüller.[59] There are thus attached to this disease the names of two Americans and one German. It has been put in a class with Gaucher's disease and Niemann-Pick's disease, all three being looked upon as disturbances of the lipid metabolism.

J. Ramsay Hunt was a neurologist of note in New York City who died all too soon. His name has become identified with a syndrome which he[60] described under the name of herpetic inflammation of the geniculate ganglion. The syndrome presents itself in three forms: herpes zoster auricularis; herpes zoster in any of the zoster zones of the cephalic extremity (herpes auricularis, herpes facialis and herpes occipitocollaris) with facial palsy; and herpes zoster of the cephalic extremity with facial palsy and auditory symptoms (tinnitus, deafness, vertigo, vomiting, nystagmus and disturbances of equilibrium). The basis of the observation of Hunt's syndrome is the fact that the facial nerve is a mixed nerve having a sensory ganglion, a cutaneous representation and a zoster zone.

In 1910 William C. MacCarty,[61] of the Mayo Clinic, made use of the term "strawberry gall bladder" as a convenient name for a pathologic condition which is frequently found in gall bladders removed surgically. At first MacCarty believed that the strawberry appearance was due to erosion of the apices of the villi of the mucosa. Moynihan[62] attributed it to minute deposits of stones. In reality the lesion is a deposit of a lipoid substance within the swollen connective tissue cells just beneath the epithelium. In a series of 5000 gall bladders removed in the course of seven years, 936, or 19 per cent, were of the

strawberry type. The strawberry gall bladder may be found with or without stones. According to MacCarty the symptoms caused by it cannot be relieved satisfactorily except by cholecystectomy.

In 1910 Nathan E. Brill,[64] of New York, described an obscure, acute infectious disease of unknown origin and unknown pathology, characterized by a short incubation period (four to five days), a period of continuous fever, accompanied by intense headache, apathy and prostration, and a profuse and extensive erythematous

Nathan Edwin Brill
1860-1925

maculopapular eruption, all of about two weeks' duration, whereupon the fever abruptly ceases either by crisis within a few hours or by rapid lysis within three days, when all symptoms disappear. He was convinced that the disease had nothing to do with typhoid or paratyphoid fever, but he admitted that it might be a non-fatal and non-contagious form of typhus fever. But in the absence of any certainty he was content to call it an acute infectious disease of unknown origin. We now know that Brill's disease is really a mild form of typhus fever due to *Rickettsia prowazeki*, a parasite that with its primary host, the louse, its secondary host, the rat, and its tertiary host, man, has made history.

Rocky Mountain spotted fever, also called tick fever, black fever or blue disease, has long been known in this country as a serious malady occurring especially in Idaho and in the Bitter Root Valley of Montana. The earliest cases were observed in 1873, but the first written account was given by Surgeon-Major W. W. Wood[65] in 1896, in a report to the Surgeon-General of the United States. From data submitted to him by eight Idaho physicians he concluded that Rocky Mountain spotted fever was a distinct disease of unknown origin. In 1899 an admirable clinical account of the disease was published by E. E. Maxey.[66] The first comprehensive study was begun in 1902 by Wilson and Chowning.[67] They reached the important conclusion, afterwards confirmed by Howard T. Ricketts,[68] that the disease is transmitted by the wood tick *Dermacentor andersoni (venustus)*. The work of Wilson and Chowning, Fricks[69] and Wolbach[70] culminated in the discovery that Rocky Mountain spotted fever is due to an organism belonging to the Rickettsia group, to which they gave the name of *Dermacentroxenus rickettsii*. The bite of a single tick is sufficient to infect a person with the disease. Fortunately the ticks must feed for some time, from two to eight hours or more, to become infective.

The distribution of Rocky Mountain spotted fever is that of its carrier, the wood tick, which is found on a number of small wild animals that are also susceptible to the disease. In man the disease sets in after an incubation period of from four to eight days with a chill and high fever; on the third, fourth and fifth days a characteristic eruption appears on the skin, sometimes on the mucous membranes of the mouth and pharynx. Restlessness and insomnia are distressing symptoms. The mortality in man varies in different localities, ranging from three to seventy per cent. More cases occur in Idaho than in Montana, but the mortality is much lower in the former state. While the disease simulates a number of acute infections, its closest resemblance is to typhus fever, and where the two are endemic the differentiation can only be made by animal experiments and cross-agglutination tests. In recent years the disease has traveled eastward, cases having been reported from Massachusetts, Pennsylvania, New Jersey, and other Eastern states.

Tularemia, a typically American discovery, has an interesting history. In 1904 a fifteen-year-old boy during a period of illness wrote to his sister that he thought his disease was due to contact with wild rabbits which he had killed. Three years later Ancil Martin,[71] an ophthalmic surgeon of Phoenix, Arizona, in a letter to Frederick G. Novy, of Ann Arbor, Michigan, reported five cases of a disease which he attributed to an affection resulting from the skinning and dressing of jack rabbits. R. A. Pearse,[72] of Brigham City, Utah,

in 1910 described six cases of a disease known among the ranchers as deer-fly fever. This article constitutes the first published report of the clinical manifestations of tularemia.

From 1908 to 1911 George W. McCoy, later director of the Hygienic Laboratory at Washington, was detailed to San Francisco as director of the United States Public Health Service plague laboratory. His duties were concerned with the examination of rats and ground squirrels for evidence of bubonic plague. When suspicious lesions were found, guinea pigs were inoculated. McCoy[73] soon discovered that the *Bacterium pestis* could not be recovered from the lesions of some of the guinea pigs and he recognized that while the disease in the guinea pigs resembled plague, it was not due to *B. pestis,* and he called it a plague-like disease of rodents. In 1912 he and Chapin[74] isolated and cultivated the organism of the plague-like disease of the California ground squirrels and called it *Bacterium tularense,* after Tulare County, California, in which the discovery had been made.

In September, 1912, William B. Wherry, professor of bacteriology at the University of Cincinnati, and Benjamin H. Lamb,[75] his assistant, described the first case occurring in man of *Bacterium tularense* infection in which the diagnosis was established bacteriologically. In this patient, a twenty-eight-year-old meat-cutter, the disease started in the eye.

Early in 1919 Theodore B. Beatty, state health commissioner of Utah, wrote to the Surgeon General of the United States Public Health Service, urging him to send one or more of his staff to investigate a disease which was incapacitating many ranchers and farmers and was called deer-fly fever. Edward Francis was assigned to this duty. In a combination garage and coal shed Francis established a field laboratory. The first patient was a fifty-two-year-old rancher who had been sick for four days following a deer-fly bite on the right side of the neck. From this patient's blood two guinea pigs and two rabbits were inoculated. All were dead on the fifth day. Autopsy revealed multiple whitish foci of necrosis of the spleen and liver. Francis[76] recognized the similarity of the lesions to those described by McCoy in his guinea pigs. The ranchman died on the twenty-sixth day of his illness. The spleen was enlarged and studded over the entire surface and throughout the pulp with tiny white spots.

Francis recovered *B. tularense* from guinea pigs which had been inoculated with splenic tissue. Five days after the rancher had succumbed to his illness, Francis himself fell ill. Despite the advice of his colleague, he took his inoculated animals back to the Hygienic Laboratory in Washington, where skillful workers kept the organisms virulent by repeated animal passages, while Francis went to the Emergency Hospital.

The work of Francis, a monument to conscientious research, demonstrated conclusively the nature of tularemia and its source, as well as the biology of *B. tularense.* The organism is able to penetrate the unbroken skin, and is conveyed by an unusual variety of insect carriers — the deer fly, the mouse louse, the rabbit louse, the rabbit tick and several varieties of wood tick.

Serological studies of tularemia in man have led to two important discoveries: first, the permanence of serum agglutinins in long-recovered cases; second, the frequent cross-agglutination of the organisms of undulant fever by tularemic antiserum.

I have gone to some length into the history of tularemia because it is from beginning to end a purely American discovery, and is another illustration of the fact already mentioned in connection with Rocky Mountain spotted fever: that work of the highest character has been done in the field under adverse conditions by scientists in government employ.

Another disease the discovery of which we owe to surgeons in the United States Public Health Service is the so-called benign lymphocytic choriomeningitis (acute aseptic meningitis). This work was done by Charles Armstrong, surgeon in the Public Health Service, and Paul F. Dickens,[77] lieutenant commander, Medical Corps, United States Navy. The disease, which appears to be increasing in frequency, at times occurring in small epidemic outbreaks, resembles bacterial meningitis. The spinal fluid, however, is sterile so far as bacteria are concerned. Through the work of Armstrong and Lillie[78] it has been proved that the cause of the disease is a filterable virus similar to that isolated by Traub[79] from white mice and by Scott and Rivers[80] from human patients. The blood serum of patients who have recovered from acute aseptic meningitis protects animals from the virus.

Haverhill fever is the name given to a febrile disease that occurred in Haverhill, Massachusetts, and was also observed in Chester, Pennsylvania. The first description was given in 1926 by Place, Sutton and Willner,[81] who called the condition erythema arthriticum epidemicum. The Chester epidemic, which occurred a year earlier and affected nearly 400 persons, was studied by Armstrong.[82] The disease has been without fatality and appears to be disseminated by food, probably milk. In the Haverhill studies an organism was found by Parker and Hudson[83] which was considered the cause of the disease. The principal features of the affec

tion have been summed up in an article by Locke and Hazard.[84]

To Burrill B. Crohn,[85] of New York, belongs the credit of having focused attention on a peculiar and important chronic inflammation of the small intestine, especially the ileum. Crohn designated the condition regional or terminal ileitis. The disease is usually a chronic one, characterized by mild abdominal cramps, moderate diarrhea, subfebrile temperature, a slowly progressive loss of weight and an advancing anemia. If not arrested it will in time involve the colon, producing the well-known picture of ulcerative colitis.

In 1932 Henry K. Pancoast[86] described a clinical syndrome which apparently had not been previously recorded. It thus becomes an American contribution to nosography. The condition is called superior pulmonary-sulcus tumor.* The tumor appears at the thoracic inlet, and is distinguished from the so-called apical chest tumor by the fact that it does not seem to originate from the lung, pleura, ribs or mediastinum. The symptoms are severe neuritis-like pain, Horner's syndrome, — contracted pupil, exophthalmos, ptosis of the lid, unilateral absence of sweating, — and the presence of a tumor on physical and x-ray examinations.

Under the name of Libman-Sacks's disease, the American literature contains a description of an affection which its discoverers[88] have designated atypical verrucous endocarditis. The disease is usually associated with lupus erythematosus, sometimes the skin lesions, sometimes the endocardial lesions predominating. The pathologic picture as demonstrated by the late Louis Gross is quite characteristic.

To Libman and his colleague Ottenberg[89] we owe an original observation on hereditary hemoptysis. There are a number of conditions apparently running in families that are characterized by spontaneous hemorrhage. Several have been differentiated, and the end is not yet. One of the last to be added to the lengthening category is hereditary hemorrhagic telangiectatic hematemesis, to which attention was apparently first called by Hyman I. Goldstein,[90] of Camden, New Jersey.

In the field of the endocrines Americans have made many important original contributions. A very significant one is by Harvey Cushing. In 1912 and more fully in 1932, Cushing[91] called attention to a definite disease syndrome which he designated as basophil adenoma of the pituitary body. The syndrome which, until Cushing's elaborate study, was attributed to a polyglandular disturbance of corticoadrenal origin, and which appears to be due to a basophil adenoma of the anterior pituitary body, is characterized by plethoric adiposity affecting the face, neck and trunk, genital dystrophy, osteoporosis and vascular hypertension. There is often early masculinization of the female patient and sexual precocity in children of either sex. While these last features, as in bearded women, are often associated with hyperplasias or tumors of the suprarenal gland of one sort or another, they may be due to basophil adenoma of the pituitary body, in the absence of apparent alteration of the adrenal cortex other than a secondary hyperplasia. Cushing has called attention to the possible relation of pituitary basophilism to essential hypertension — a significant lead in an obscure field.

Peritoneal endometriosis, a condition which gynecologists consider a definite clinical entity, was brought to the attention of the profession by John A. Sampson,[92] of Albany, New York. The disease arises from implantation of endometrial tissue disseminated through the escape of menstrual blood into the peritoneal cavity.

Hyperfunctioning adenomatous goiter (toxic adenoma) was proposed as a clinical entity by Henry S. Plummer,[93] of Rochester, Minnesota, who maintained that it differed from exophthalmic goiter in characteristic ways. This opinion is not shared by all writers on hyperthyroidism; nevertheless, it is proper that Plummer's syndrome should find a place among the American contributions to nosography. Patients presenting the symptoms of hyperfunctioning adenoma have as a rule had a nodular goiter for many years. Usually without apparent cause, sometimes after prolonged administration of iodine, hyperthyroid symptoms appear, generally without much exophthalmos. Hypertension is fairly frequent. Unless the heart has been seriously damaged, operation is nearly always followed by the disappearance of the thyrotoxic symptoms.

Hyperinsulinism owes its identification to Seale Harris,[94] of Birmingham, Alabama, who first called attention to it in 1924. Hyperinsulinism leads to hypoglycemia, and this in turn produces symptoms comparable to those following an overdose of insulin. In some cases a tumor of the pancreas consisting of islet tissue has been found. Cures have been achieved by the removal of such tumors.

Meigs's tumor is the name given to a recently described syndrome which appears to be a definite clinical entity, even if somewhat obscure. It is essentially a fibroma of the ovary with hydrothorax and at times with ascites. Scattered reports have appeared in the literature, but the paper that brought the subject definitely before

*Recently the idea that the superior pulmonary-sulcus tumor is a clinical entity has been called in question.[87]

the profession was that by Joe V. Meigs and John W. Cass.[95, 96] The condition derives its importance from the fact that despite the presence of fluid in the chest and in the abdomen, the ovarian tumor may be a fibroma and hence benign; furthermore, the removal of the tumor usually causes the disappearance of the hydrothorax and the ascites.

Time does not permit me to include in this account all the diseases discovered or first clearly defined by Americans. I shall therefore bring this essay to a close. In concluding, I have the honor to bring to you the official greetings of the College of Physicians of Philadelphia. Our college, which is but six years younger than your own venerable society, is not a teaching institution but a medical society and library. It has a romantic interest in the Shattuck Lecture for the reason that the founder, George Cheyne Shattuck, was an associate fellow of the college. His distinguished son was likewise an associate fellow. On the death of George Cheyne Shattuck, the younger, the college requested Dr. Alfred Stillé, who had been Shattuck's companion in Paris, to deliver a memorial address, a tribute rarely paid to the memory of an associate fellow of the college.

1520 Spruce Street.

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CLOSED INTRAPLEURAL PNEUMONOLYSIS WITH THE ONE-PIECE OPERATING THORACOSCOPE*

LOUIS RABINOWITZ, M.D.,† AND EDWARD J. ROGERS, M.D.‡

PITTSFORD, VERMONT

CLOSED intrapleural pneumonolysis is the procedure whereby certain types of pleural adhesions in induced cases of pneumothorax are divided under endoscopic control. Experience has shown that about 50 per cent of all pneumothorax therapy is rendered ineffective by the presence of pleural adhesions; as a result, the pulmonary lesion fails to collapse adequately, and tubercle bacilli are still found in the sputum three to six months after the induction of the pneumothorax.

For intrapleural pneumonolysis we have selected 54 patients in whom pleural adhesions appeared to be the cause of the persistent presence of tubercle bacilli in the sputum, and in whom the existing pulmonary cavities had failed to close. Seventy operations were performed on these patients.

For the procedure of closed intrapleural pneumonolysis a sufficiently large pneumothorax is essential. The adhesions to be severed must be attached close to the diseased lung area, and should be more than 2 cm. long. They should be taut, and should contain no large blood vessels or lung tissue. Serous or purulent exudates, or contralateral lesions, offer no contraindication to this procedure.

Closed intrapleural pneumonolysis is effected by two types of apparatus. The most frequently employed procedure utilizes one intercostal space for the pleural endoscope or thoracoscope, while the cautery or high-frequency-current electrode enters the chest cavity through another intercostal space. A more infrequent technic, and the one used in this series, is that wherein the thoracoscope and the high-frequency-current electrode enter through the same intercostal space.

Several one-piece instruments have been devised, namely those of Hervé, Singer, Davidson, Chandler, Kornitzer, Maendl, Pinchin and Morlock, and Cutler.

*From the Department of Medicine, University of Vermont College of Medicine, Burlington, and the Vermont Sanatorium, Pittsford.

†Instructor in clinical medicine, University of Vermont College of Medicine; associate medical director, Vermont Sanatorium.

‡Assistant professor of medicine, University of Vermont College of Medicine; superintendent and medical director, Vermont Sanatorium.

DESCRIPTION OF INSTRUMENTS

The principal instruments used in our cases of intrapleural pneumonolysis were the Cutler operating forceps thoracoscope and the utility carrier with its accessory instruments. The operating forceps thoracoscope consists of a telescope with a forward and oblique field of vision. The center ray in the field forms an angle of about 27 degrees with the telescopic axis. By rotating the telescope one may double the angle of vision, and increase the field of vision fourfold. A 3-volt bulb is attached in front of the objective lens and is activated by a connecting cord with a regulating switch near the eyepiece. A battery handle and hook, with two regular flashlight cells, supplies the current for the telescope bulb. A built-in rotary rheostat gives a means of regulation.

The operating forceps consists of serrated jaws worked by handles. The upper or stationary jaw contains an insulated bushing in which rests the cutting electrode. A small ball at the end of the latter aids in orientation. Both jaws and electrodes are always within the field of vision. Near the handle is a terminal into which is inserted the power cord.

The telescope can be released by turning a thumbscrew and placed in the utility carrier. The latter instrument allows the rapid exchange of various types of electrodes as needed. The most commonly used types are the longitudinal and lateral curved cutting electrodes, and rarely the coagulating ball electrode. These electrodes always appear in the upper third of the telescopic field of vision. The longitudinal and lateral curved cutting electrodes are useful in cutting and dissecting short adhesions. The ball electrode can be used for coagulating bleeding surfaces.

The cannula, through which operating forceps and utility carrier are introduced into the pleural cavity, is 26 French in caliber and 20 cm. long. It has an adjustable collar which regulates the depth of projection into the chest of the trocar, which can be locked into place around the cannula. The trocar can be removed and the cannula opening closed by an air plug at any time. The current used is supplied by a single vacuum-tube unit, which is activated by a foot switch.

OPERATING METHOD

X-ray films and fluoroscopic study of the patient's chest often aid in localizing the adhesions which are preventing cavity closure, but almost as often only thoracoscopic examination is of value. Too much reliance must not be placed on x-ray study. Since more than 90 per cent of the adhesions that require lysis in the pleural cavity are found in the upper posterior quadrant of the chest, the location of the point of entry must be anterior. One should enter through the intercostal space that will bring the electrode not closer than 10 cm. to the adhesion; otherwise the degree of instrumental manipulation is very limited. If a pleural effusion is present, and if it seems of sufficient quantity to obscure the working area, it should be removed as completely as possible the day before operation. Unless the intrapleural pressure is negative, a pneumothorax refill before the operation is unnecessary. Preoperative medication is but rarely needed.

On the morning of the operation all the instruments and lighting cord are submerged in a 1:1000 solution of mercury oxycyanide for thirty minutes, and then left in a tray of sterile water for ten minutes. The instruments used for local anesthesia are boiled.

The patient is placed on the operating table on his back, with the upper half of the trunk elevated about 5 degrees. The half of the chest to be operated on is painted with dilute tincture of iodine, and is draped with three towels and a laparotomy sheet. The face is directed to the opposite side.

Infiltration anesthesia is invariably used. A 1 per cent novocain solution containing adrenalin hydrochloride is satisfactory. A skin wheal is produced over the intercostal space selected. A 19-gauge needle is introduced into the pleural cavity to a point where suction with a syringe produces air bubbles. The needle is then withdrawn slowly until no air is obtained, and about 10 cc. of solution is injected. About 5 cc. more is injected along the needle track. The cannula collar is adjusted so that the trocar point cannot penetrate beyond the distance determined by the anesthesia needle as the parietal pleura.

A 1-cm. incision is made parallel with the ribs, and the trocar and cannula are introduced with a rotary motion through the tissues. The trocar is removed and the cannula is further introduced until a characteristic blowing sound is noted. The utility carrier, with the telescope and longitudinal electrode in place, is then introduced through the cannula, the lamp brightness having been adjusted.

The collapsed lung is usually first seen as a moving, bluish-gray mass. The adhesions are white, yellow or pink. The intercostal, internal mammary and innominate veins are often recognized. Frequently the phrenic nerve can be seen. The adhesions are of many types and combinations — fresh arachnoid adhesions, as well as the tougher round and flat string adhesions. Cord and band adhesions containing blood vessels may be

seen. Frequently, an adhesion which by x-ray examination appeared to be a simple fibrous band proves to be a funnel, spool or diffuse adhesion containing lung tissue.

If the adhesions are of the string, cord or band type, they may be grasped by the forceps close to the chest wall. The instrument is passed through the cannula with the telescope in place. The field of vision is decreased, but, the site having been studied beforehand, no difficulty is experienced. The adhesion is grasped very lightly. The degree of vascularity may be noted by pressure application. The current is started, and there is immediately noted a white zone about 0.3 cm. wide on each side of the electrode. Within thirty seconds,

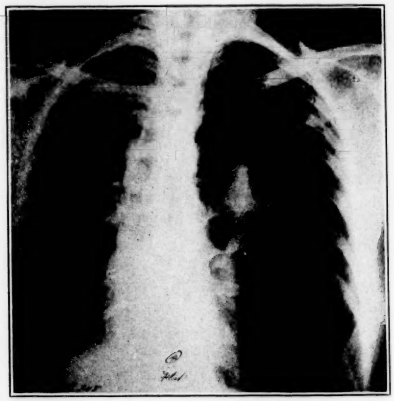

Figure 1. *Case 1. Artificial pneumothorax induced six months previously. Sputum contained tubercle bacilli and cavity held open by thick band adhesion.*

there is usually seen a parting of the tissue on each side of the electrode, and the adhesion gives way with a snap (Figs. 1 and 2).

Short adhesions of the triangular, spool or diffuse type require dissection. The utility carrier, containing the longitudinal or lateral curved electrode, is used. In most cases the area to be dissected is first demarked by a zone of coagulation. The point of the electrode is next pushed into the nearest edge of the adhesion and the current is turned on, then off. With the current off, the point of the electrode at once becomes cold. The electrode is then used to dissect the adhesion fibers bluntly from the parietal pleura. When another edge is seen the current is turned on, then off, and the cold dissection is repeated. Sometimes a period of one month is permitted to elapse after the first dissection; considerable stretching may then occur, and the remainder of the adhesion can conveniently be severed (Figs. 3 and 4). Oozing can be controlled by touching the electrode to the bleeding area or by burying it as close to the chest wall as possible, and employing the coagulation current.

At the conclusion of the operation the pleural cavity is inspected and the instruments are with-

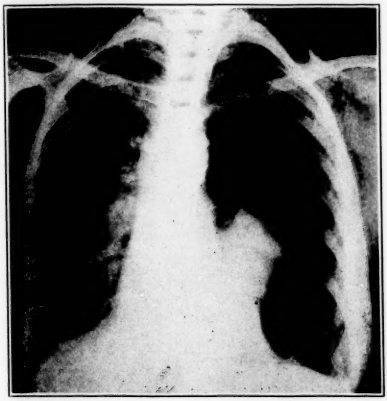

Figure 2. *Case 1. Adhesion grasped by operating thoracoscopic forceps and severed in twenty-five seconds. Cavity closed and sputum free of tubercle bacilli during week following the procedure.*

drawn. The incision is closed with a mattress suture of medium-sized silkworm gut. Sterile gauze and two strips of adhesive are sufficient as a dressing. The patient remains on his back in a semi-recumbent position for twenty-four hours and is cautioned not to cough or talk. At the end of this time the chest is fluoroscoped. If there seems to be no loss of pneumothorax no refill is given until forty-eight hours have elapsed, thus often avoiding a temperature reaction that may occur following an earlier refill.

At the end of twenty-four hours after operation the patient may be allowed lavatory privileges. The suture is removed forty-eight hours after operation. He should remain on modified bed rest for one week.

No nerve injuries, such as the resultant ulnar neuritis described by Schröder, were noted in our series of 70 operations among 54 patients.

There was no considerable skin emphysema, or loss of pneumothorax such as that described by Moore in 0.29 per cent of the 2043 cases he collected.

Serious hemorrhage, which was noted in

0.73 per cent of Moore's cases, in 2 per cent of the 88 cases of Peters and Cornish, and in none of Alexander's 111 or Welles's 141 cases, was absent.

There were no pleurocutaneous sinus complications such as described in 3.6 per cent of Anderson and Alexander's cases and in 0.14 per cent of Moore's cases.

Mixed empyema was absent. This was a complicating feature in 1.8 per cent of Moore's cases, in 1.8 per cent of Anderson and Alexander's cases,

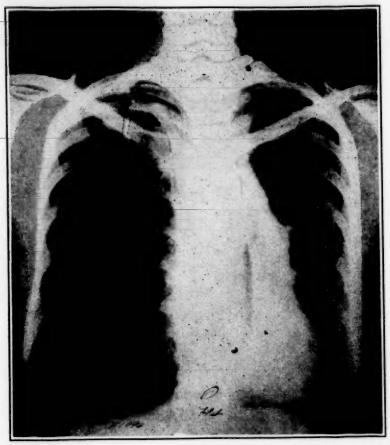

Figure 3. *Case 2. Artificial pneumothorax induced eight months previously. Sputum contained tubercle bacilli and cavity held open by a diffuse adhesion containing lung tissue.*

in 1.4 per cent of Welles's cases, in 1.3 per cent of Maurer's 160 cases, in 3.0 per cent of the bilateral and 0.3 per cent of the unilateral cases from a group of 250 reported by Coryllos and in none of Drash's 230 cases. The last-named used the Davidson instrument.

Tuberculous empyema occurred in 1 (2 per cent) of our series. It disappeared following several aspirations, and no effusion has reappeared after more than one year. This complication was present in 8.07 per cent of the Moore series, in 3.6 per cent of the cases reported by Anderson and Alexander, and in 26 per cent of the 136 cases of Matson where a cautery was used and in 3 per cent of his 78 cases where high-frequency current was employed. Drash reported this complication in 2.6 per cent of his series, and Coryllos noted it in 2.5 per cent.

Serous exudate was seen in 8 (15 per cent) of our series before operation. It was noted in 12 cases (22 per cent) of the series after operation, so that 4 cases (7 per cent) had this complication as a direct result of operation. Drash reported it as a complication in 6 per cent of his cases.

No deaths could be ascribed to the intrapleural pneumonolysis operation.

RESULTS

By means of intrapleural pneumonolysis Anderson and Alexander were able to achieve closed cavities and a negative sputum in 63 per cent of their cases, Matson in 69 per cent and Coryllos in 70 per cent of his unilateral and 25 per cent of his bilateral group. Drash was successful in 75 per cent of his cases.

Of the 54 patients in our series, 30 had unilateral and 24 had bilateral involvement. A single-stage operation was performed in 38 cases, while the

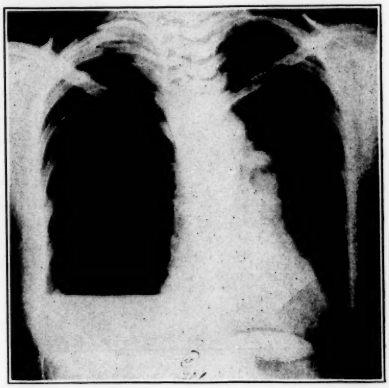

Figure 4. *Case 2. Diffuse adhesion dissected by longitudinal cutting electrode from chest wall in two stages, totaling seventy-five minutes of operating time, two months apart. Cavity closed and sputum free of tubercle bacilli during month following the second procedure.*

two-stage procedure was used in 16 cases. The pre-existing pulmonary cavities were closed and the sputum became negative for tubercle bacilli in 26 (87 per cent) of the unilateral cases and in 10 (42 per cent) of the bilateral cases. The total number of cases in which the sputum became negative was 36 (67 per cent).

DISCUSSION

Closed intrapleural pneumonolysis is a useful procedure and can be employed in about 20 per cent of all induced pneumothorax cases where pleural adhesions prevent effective collapse.

The one-piece instrument, which permits the

severance of adhesions through the same intercostal space, is efficient. Utilizing this instrument in our series, no appreciable emphysema or loss of pneumothorax could be noted. The cutting depth appeared small and coagulation certain. Smoke was never a factor. With the current off the electrode almost immediately became cold enough for blunt dissection.

The apparatus is not complicated. The field of vision was limited during cutting with the forceps, but with previous study a large field was unnecessary at the operating stage. The instrument has always been found to be flexible. Desiccation, coagulation and cutting are almost automatic. The initial cost of the apparatus, where the power unit is not already present, is several times that of the two-piece cautery outfit.

The end results appear to be as good as those obtained with the two-piece apparatus.

SUMMARY

The technic of the use of the one-piece operating apparatus in closed intrapleural pneumonolysis is described.

As a result of operation on 54 patients the sputum became negative and the cavities closed in 67 per cent.

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PHYSIOLOGIC DULLNESS OF THE RIGHT APEX

Summary of Opinions

ROBERT W. BUCK, M.D.*

BOSTON

TODAY, if we want to know whether or not a person has pulmonary tuberculosis we x-ray his chest. To say that many patients cannot afford this luxury or that they live in remote districts too far away from good x-ray equipment is only an excuse, and not a good one, for the lack of medical care. Perhaps the chief present value of physical examination in connection with chronic diseases of the chest lies in the discovery of abnormalities that should have been recognized long before, but which because of neglect were allowed to reach a stage where the intensified signs of their presence are detectable even by the unaided senses of the examiner. The question, "How far are the external means of exploring the condition of the internal organs to be considered useful and important in medical practice?" which was offered to Boylston Prize Essay contenders somewhat more than a hundred years ago, is still pertinent, but even Dr. Oliver Wendell Holmes[1] would have to give a different answer today than that which won him a fifty-dollar gold medal in 1836.

During the intervening century, and before the final perfection of x-ray photography, a considerable amount of study, investigation and discussion was devoted to the finer points of physical diagnosis. A residual portion of the knowledge thus acquired, although not of uniform quality, is still taught to medical students early in their course of instruction. It is at least of scholarly or historical interest to look into the origin of some of these principles now widely held. Among them is the conception that the apex of the right lung evinces a certain physiologic dullness not apparent on percussion of the left lung.

Strangely enough, the existence of this physiologic dullness remains known almost exclusively to American physicians. By physiologic dullness I do not mean the dullness of well-developed shoulder muscles in right-handed laboring men; I mean that less tangible dullness which is, in the opinion of nearly every American textbook-writer who stresses it, "not sufficiently emphasized."

Who first discovered it? I can only guess. Auenbrugger†[2] knew it not in 1761, nor Collin[3]

*Assistant professor of preventive medicine and instructor in physical diagnosis, Tufts College Medical School; physician, New England Medical Center.

†"The left side yields this sound [resonance] from the clavicle to the fourth rib, anteriorly, and in the back and laterally, in the same extent as the other side." *Inventum Novum*, Obs. I, § iii. 2.

in 1824, nor Piorry[4] in 1828 or 1866. But in 1836, William W. Gerhard,[5] physician to the Blockley Hospital of Philadelphia, observed — to the edification of young Holmes among others:

> Percussion must be made above, upon, and immediately below each clavicle. In these places a very slight deviation from the natural standard may be detected. It should, however, be recollected, that a very small difference in favour of the left side does not indicate induration of the right lung, as the summit of the left is naturally a little more sonorous.

Skoda[6] (1844) gave no indication that he knew of this small difference. Mailliot[7] (1843) found that percussion of the chest gave "les mêmes résultats d'un côté comme de l'autre, depuis le sommet des poumons jusqu'à la quatrième côté." Henry I. Bowditch[8] (1846) told the young stethoscopist: "The tops of the shoulders . . . are very important when compared together. The slightest *difference of note,* combined with other physical and rational signs, will perhaps decide your diagnosis"; and he nowhere qualified this issue — or confused it — by reference to any physiologic difference.

Ten years later Austin Flint,[8] who discovered so many things, concluded:

> In the majority of persons in health having well-formed chests, there is not an absolute equality in the resonance existing at the summit of the chest in front on the two sides. It appears also, that, as a general rule, the disparity consists in a greater degree of resonance, more vesicular quality, and relatively, lowness of pitch, on the left side . . . the disparity exists in cases in which there is no apparent difference in the muscular covering, in this situation.

This conclusion was based on the examination of 20 presumably normal subjects, of whom 8 showed a percussion sound in all respects equal on the two sides, and 10 a greater degree of resonance on the left side. In the same group percussion of the apices posteriorly showed no disparity in 13, and diminished resonance on the right in 4. Flint is the only author who, so far as my reading goes, presents any experimental evidence in support of his contention, and it must be admitted that this evidence is hardly incontrovertible.

In 1866 Gerhardt[10] made the nearest approach to recognition of the phenomenon ever achieved by a German physician. "The inner portion of the infraclavicular region gives the clearest note of any part of the chest, and here again the left side frequently gives a clearer note than the right (lesser development of musculature)." No other German, to my knowledge, has verified this observation, and the parenthetical explanation might be taken as evidence that Gerhardt himself was only noting an occasional superficial and rather obvious difference.

Disappointingly, Loomis[11] (1872) distinctly stated that any difference in the percussion note in the infraclavicular region on the two sides was important. The same assertion was implied in the well-known British text of Gee[12] (1870), and in the equally celebrated French manual by Barth and Roger[13] (1870). Eight years later, however, Delafield and Stillman[14] reiterated that "on the right side, in front, from the lower edge of the clavicle to the top of the fourth or fifth rib, there is pulmonary resonance, usually of higher pitch than that on the left." But in 1882 Cutler and Garland,[15] of Boston, make no mention of the finding. Neither did the British author Anderson[16] in 1889, and in 1891 Gibson and Russell,[17] of Glasgow and Edinburgh, only say hesitatingly that "slight or comparative dulness is frequently present at one or other apex, more commonly at the right. It is sometimes regarded as physiological, but this is doubtful." In 1890 Shattuck[18] did not mention physiologic dullness in his book *Auscultation and Percussion.* Cabot,[19] however, in 1900 clearly and unequivocally stated, as "a very important point and one not sufficiently appreciated by students," that "the apex of the normal right lung is distinctly less resonant than the apex of the left in a corresponding position."

Dr. Cabot's book found immediate favor, and has ever since remained the most widely used American textbook of physical diagnosis. From 1900 on there is almost universal agreement among American observers. Thus, Le Fevre[20] (1902) wrote: "The percussion note over the right lung, from the apex to the second interspace in the axillary line, is slightly higher in pitch and duller than on the left side." Anders[21] (1907) said: "The apex of the *right* lung [percussed in the supra-clavicular space] is not so resonant as the left." DaCosta[22] (1908) asserted: "The percussion sound is somewhat higher pitched and less typically vesicular in the right than in the left infraclavicular area. To a minor degree these differences are also found in the supraclavicular regions." Norris and Landis[23] (1918) said: "The percussion note from the right upper apex to the second interspace is slightly higher-pitched, less resonant, and at times has a slightly tympanitic quality," and they gave five good reasons why this should be so. Rose[24] (1921) stated: "The percussion note as elicited by percussion over the right apex is normally of slightly higher pitch than the note which is elicited over the opposite apex." Pratt and Bushnell[25] (1925) pointed out: "The percussion-note . . . of the right apex is often shorter and higher pitched than that of the left apex." Major[26] (1937) found himself in agreement: "The percussion note of the right apex is often shorter and higher pitched than that of the left apex. This fact, often insufficiently emphasized, should always be kept in mind." It is with

some astonishment then, that we read in Sutton's[27] (1937) text: "It is important to remember that the summit of the right apex is normally more hyperresonant than that of the left." But after all, one can hardly expect that teachers will go on repeating the same thing indefinitely. Change is of the essence of progress.

The present summary is intended to be primarily a discussion of the development of an idea. It may not be amiss, however, to consider what experimental evidence is available on the subject.

Dr. Richard Cabot[28] writes: "I don't know how the question can be settled until an automatic percussion-apparatus and an automatic record of its sounds are devised. This could be done, I fancy."

It has indeed been done, though not with the primary purpose of comparing the percussion notes of the two apices. I refer curious students to two excellent sources: Edens's *Lehrbuch der Perkussion und Auskultation* (Berlin: Julius Springer, 1920) and Martini's monograph *Studien über Perkussion und Auskultation* (Leipzig: F. C. W. Vogel, 1922). From these it may be learned that a progressive development of methods for the graphic recording of the auditory phenomena of percussion and auscultation has occurred in Germany during the past seventy-five years. In 1860 Seitz and Zamminer[29] indicated the desirability of such methods, and in 1862 Gerhardt[30] obtained graphic records by means of a sensitive flame. It was not until 1907 that Selling[31] at the instigation of Friedrich Mueller set himself to the task of clarifying the obscure subject of percussion. Data of interest bearing on the matter were successively supplied by Moritz and Röhl,[32] Geigel,[33] and Edens and Ewald.[34] The completest and most accurate development of graphic registration was that of Martini, based largely on the preliminary work of Edens.

Martini sought, apparently successfully, to substitute for subjective observation an entirely objective method of recording. Making full use of the earlier studies in the physics of transmitted sound by Helmholtz, Lord Rayleigh, Wien and Frank, he collected an abundance of data, much of it highly technical and of theoretical interest. A convenient summary of his clinically important conclusions is available in the English translation by Farber[35] of his elementary textbook, and reference is made to some of them in an article by Martini[36] which appeared some years ago in the American literature.

So far as the matter of physiologic dullness of the right apex is concerned, the conclusion is reached and stated by Martini that when all proper precautions are observed as to the position of examiner and patient, choice of symmetrical points for percussion, and so on, *any difference in sound between the two sides is always of pathological significance unless explainable by an anomaly of the chest wall.* Backed by a great deal of experimental evidence, this conclusion seems to me to be impressive and hardly to be disregarded.

* * *

Reference to original sources seems to indicate that the inventors and early careful observers of the art of percussion did not recognize the existence of physiologic dullness of the right apex, that physiologic dullness is a concept of American clinicians not based upon or justified by objective experimental results and that one must seriously doubt that it exists.

5 Bay State Road.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24421

PRESENTATION OF CASE

A thirty-seven-year-old American housewife entered the hospital complaining of jaundice, weakness and swollen abdomen and ankles.

Three years before entry the patient gave birth to her third child. All three pregnancies and deliveries were essentially normal. Sixteen days after the last delivery her neck became swollen, red and stiff and this was followed by migratory arthritis involving her back, ankles, carpophalangeal joints, thumbs, wrists, elbows and both shoulders. A friend gave her some pills which she took until six months before entry, but the nature of these could not be ascertained. So far as could be determined she had not taken these pills during the past six months. Five months after the onset of the arthritis she had all of her teeth extracted but had remissions of arthritis from time to time afterward. She had no bed rest, but received vitamin B, iron, heat, massage, violet ray, high-carbohydrate diet and "injections." Four months before entry her joints improved, leaving only a stiff left elbow and right shoulder. About this time she noted a feeling of nausea on rising in the morning, which increased within a few days and resulted in vomiting. She had a sense of constriction of the neck and of strong pulsations in the blood vessels, palpitation, paresthesia in all extremities and a transient "prickly-heat" rash which at times lasted all day. She was listless, "felt as if she were going to die" and wanted to lie down at all times. There was questionable fever but no chill. Vomiting relieved her to some extent but left her "no good for the rest of the day." Coincidentally there were sharp lancinating pains which radiated across her back leaving both infrascapular regions very painful. These attacks were precipitated by the sight or intake of food, lasted five minutes to an hour and were relieved by vomiting. Three months previous to the onset of nausea the patient drank a quart of milk a day. Since the onset of her symptoms, however, she could not tolerate milk, eggs or fried foods. Later she noted clay-colored stools and an increasingly dark urine. Three weeks prior to entry she felt still weaker and noticed the onset of jaundice. She continued to vomit brownish colored material, but there was no increase in the frequency or severity of the attacks. Two weeks before entry she noted for the first time an increase in the size of her abdomen and legs. Pruritus developed, and she had a "dopey" feeling which made it difficult for her to keep her eyes open. A week and a half before entry she entered an outside hospital where she was treated with intravenous glucose. During the week before entry the size of her abdomen and legs rapidly increased.

She had never been jaundiced previously, and there was no family history of jaundice. Her father died of carcinoma of the esophagus, and her mother of "cancer" of the leg. Both the patient and her mother had had psoriasis and arthritis.

Physical examination showed a well-developed woman, who had apparently lost weight recently. The skin, sclerae and mucous membranes were deeply jaundiced. There were patches of psoriasis below the knees, elbows, under the right breast and on the scalp. Examination of the chest showed diminished excursion of the diaphragm. There were diminished breath sounds and fremitus over both bases and a few rales. Examination of the heart was negative. The blood pressure was 121 systolic, 80 diastolic. The abdomen was very greatly distended, with extrusion of the umbilicus. There was tympany anteriorly and shifting dullness in the flanks. The liver was questionably ballotable 3 cm. below the costal margin. There was tenderness beneath the costal margin in both flanks. Pitting edema was present over the ankles and sacrum. The stools were light yellow in color.

The temperature was 98.6°F., the pulse 78. The respirations were 30.

Examination of the urine showed a 4+ bile test and an occasional white blood cell in the sediment. The blood examination showed a red-cell count of 4,040,000 with 80 per cent hemoglobin and 1.5 per cent reticulocytes. The white cell count was 9050, 70 per cent polymorphonuclears. A van den Bergh was biphasic with 16.5 mg. per cent bilirubin. The nonprotein nitrogen of the blood serum was 16 mg. per cent, the cholesterol 88 mg. A Takata-Ara test showed a 000++0 reaction. A bromsulfalein test for liver function showed 100 per cent retention in five minutes and 90 per cent retention in thirty minutes. A blood Hinton test was positive, a Wassermann negative.

X-ray examination showed a negative chest except for slight thickening of the lateral pleura on the left side. The abdomen was large, but no unusual soft-tissue masses were visible. The spleen was not visible. The subdiaphragmatic portion of

the liver appeared fairly small. No gall stones were seen.

On the fourth hospital day a paracentesis yielded 3 liters of yellow bile-stained fluid, which had a specific gravity of 1.010 and contained 60 mononuclear cells and 55 red cells per cubic millimeter. The patient was on a soft, high-carbohydrate diet but on the sixth hospital day regurgitated small amounts of partially digested food. Fluid reaccumulated rapidly in the abdomen. On the eighth hospital day she was irrational and drowsy. Her temperature, though normal for the first eight days, was then gradually rising, remaining around 100°F. The pulse varied from 70 to 110, the respirations remained at 20. On the twelfth hospital day a second paracentesis yielded 4.5 liters of yellow fluid with a specific gravity of 1.008 and containing 274 red blood cells and 54 white blood cells per cubic millimeter. On the fourteenth hospital day she was still semicomatose, took food and fluids poorly, and showed progressive ascites and edema. The temperature and pulse were gradually rising. On the nineteenth day her temperature was 103°F., pulse 125, and the respirations 20. She was completely disoriented and could take nothing by mouth. On the twenty-first hospital day coarse rales developed at both lung bases. The temperature was 103.5°F., pulse 150 and respirations 25. These symptoms gradually increased and she died on the twenty-second hospital day.

Differential Diagnosis

Dr. Wyman Richardson: I take it there was no evidence of a large mass in the abdomen by x-ray and that they were not able to demonstrate an enlarged spleen.

It seems quite obvious that she died of liver failure, and before discussing the differential diagnosis, I shall speak for a moment about some of her symptoms. She had arthritis, presumably rheumatoid arthritis, and it is very interesting that at about the time of the onset of her terminal symptoms the arthritis went into a rather sensational remission. It is well known that patients with rheumatoid arthritis not infrequently get a spectacular remission in the presence of jaundice, and it has been thought that the jaundice itself was the factor involved. I believe that attempts have been made to treat rheumatoid arthritis by the use of bile preparations. I should suggest, though the idea is possibly fantastic, that it is not the jaundice but the diseased liver that is responsible, by letting some substance such as unsplit protein through into the circulation, which has some effect on the circulation of the joints.

The fact that she had psoriasis would make some people speak of psoriatic arthritis. Both are very common diseases and are often hereditary. I see no reason why a single patient could not have both diseases at the same time, and I do not feel that much is to be gained by calling it a special kind of arthritis.

She received pills up to six months before entry, that is to say up to one or two months before the onset of the terminal symptoms. As we all know, patients with arthritis are apt to take all kinds of medicine, many containing cinchophen or cinchophen derivatives. It is at least fair to say that she might have been taking large amounts of cinchophen.

She then had the onset of a curious set of symptoms which are rather difficult to explain. These were constriction of the neck, strong pulsations in the blood vessels, palpitation, paresthesia in all four extremities and a transient "prickly-heat" rash. If we exclude pernicious anemia and vitamin deficiency as a cause for paresthesias, the commonest cause is some effect on the vasomotor control of the arteries, such as in Raynaud's disease. It is possible, it seems to me, that she was becoming sensitive to some protein which the liver either did not destroy or conjugate properly, and had a definite protein reaction after eating. That might account for the rash. It might be urticarial in nature. On the basis of liver damage, I cannot account for it definitely in any other way.

One more point to be considered is the pain in the back and infrascapular region. Is that going to help us in the differential diagnosis which I shall speak of in a moment? I think it is fair to say that it is possible to get all types of pain with any type of liver disease, and one does not have to assume in the presence of pain that there are stones in the region of the biliary tract.

Comments in regard to the laboratory findings are unnecessary. I might mention the question of a biphasic van den Bergh. I do not know whether it is my ignorance or not, — it very likely is, — but it is difficult for me to lay any stress on whether a van den Bergh is direct, indirect or biphasic, and I do not believe in this case it helps the differential diagnosis to know that it is biphasic.

I am assuming that this patient died of hepatic failure. I do not see how it could be a stone in the common duct because the downward progress of the disease seems to have been too rapid and there is too much evidence of liver damage and not enough of a chronic story, even though one might assume that the pain that she had was due to stone. The history does not suggest the type of

pain we have with stone. I should hate to think that this patient had a small stone in the common duct with rapidly rising infection behind it and destruction of the liver; I do not believe she did. I rule out cancer because when cancer patients die with severe jaundice they show, in most instances, a tremendous enlargement of the liver. If she had a small carcinoma obstructing the common duct I should doubt whether she could have gone into as rapid hepatic failure as this. Such a lesion, like stone, would give a more prolonged and less stormy course. In portal cirrhosis, this degree of jaundice coming on so early is unusual; again, the course is too rapid, and there is no evidence of enlarged spleen or of esophageal varices. The course is also too short for biliary cirrhosis, so-called, which is a disease of very long standing. Furthermore, the liver in biliary cirrhosis, if my learning is correct,—I have seen very few,—is very much enlarged. I shall mention syphilis of the liver because of the positive blood Hinton test. Usually this results in a very fibrous condition with a splitting up of all lobes of the liver, the so-called hepar lobatum. Some of these abnormal lobes are often palpable. I cannot conceive of such a condition explaining the symptoms of this case.

I therefore assume that this patient had subacute atrophy of the liver on a toxic basis. As to whether cinchophen taken over a long period of time could have produced symptoms appearing a month after she stopped taking it, I am not certain. We also have to consider the injections she received and whether they might have contained either cinchophen or cinchophen derivatives or a heavy metal. It is interesting that she did not stop taking medicine or having injections until shortly before the symptoms appeared, and I think it is fair at least to assume that either cinchophen or some heavy metal was accountable for the atrophy. I should say that she had subacute atrophy of the liver and died of liver failure, and in addition had rheumatoid arthritis and psoriasis.

Clinical Diagnoses

Acute yellow atrophy.
Rheumatoid arthritis.
Psoriasis.
Catarrhal jaundice?
Carcinoma of head of pancreas?
Carcinoma of gall bladder?

Dr. Richardson's Diagnoses

Subacute atrophy of the liver.
Rheumatoid arthritis.
Psoriasis.

Anatomical Diagnoses

Subacute atrophy of the liver.
Rheumatoid arthritis.
Psoriasis.
Ascites.
Peripheral and pulmonary edema.

Pathological Discussion

Dr. Tracy B. Mallory: The liver of this patient was small, weighing only a little over 800 gm. It was intensely jaundiced and very flaccid, and in large areas all the liver cells seemed to have disappeared leaving nothing but the fibrous stroma. In other areas a moderate amount of regeneration had occurred. There is no way in which the postmortem examination can help to explain the etiology. It is interesting to speculate, as Dr. Richardson has, whether atrophy can occur as a delayed reaction after the taking of cinchophen. We have good evidence of such delayed reactions after arsenic, and it is not inconceivable in relation to cinchophen. How do you feel about that, Dr. Jones?

Dr. Chester M. Jones: The onset of symptoms in most of the cases has occurred during the administration of the cinchophen, but I can recall cases of hepatic insufficiency after arsenic and one or two after antimony and gold in which a very long period of time occurred before a hepatic reaction took place. You can have cinchophen reactions with apparent liver failure following small doses or you can have prolonged administration before anything happens.

Dr. John D. Stewart: How often do you find ascites in these cases?

Dr. Mallory: Practically every case will show it post mortem; a fair proportion will show it clinically.

Dr. Jones: A very high percentage of cases will show marked ascites, with the abdomen greatly distended.

Dr. Mallory: The older textbooks say that it does not occur, but in our experience it is almost invariable. Moreover many cases show some degree of peripheral edema, such as this one did.

CASE 24422

Presentation of Case

A sixteen-year-old, American schoolgirl entered the hospital for study of a mass in her left upper chest.

She had always been perfectly well and healthy except for fairly frequent head colds. She had had four during the previous two months, each of which was mild and lasted about ten days. One of these occurred six days before entry. She had

no fever or chest pain during the attacks but did have a hacking cough productive of small amounts of mucoid sputum. Four days before entry she fainted while skating but recovered promptly and was able to continue. Three months before entry a routine tuberculin test was positive, and two weeks before entry x-ray films of the chest showed a mass in the left upper chest. She had not been exposed to tuberculosis and had had no symptoms of it. Except for the colds she had had no cardiorespiratory symptoms. Her past and family histories were essentially negative.

Physical examination revealed a well-developed and nourished girl in apparent good health. There was moderate injection of the mucous membranes of the pharynx. The left chest showed diminished expansion and dullness from the apex down to the fourth interspace anteriorly and to the sixth interspace posteriorly. Breath sounds, voice sounds and tactile fremitus were diminished over this area. The lungs elsewhere were negative, and there was no apparent shifting of the mediastinum. The heart was negative, and the blood pressure was 120 systolic, 80 diastolic. The abdomen showed no abnormalities. Pelvic examination was not done.

The temperature was 97.8°F., the pulse 90. The respirations were 20.

The urine examination was negative. The blood showed a red-cell count of 5,400,000 with 90 per cent hemoglobin, and a white-cell count of 8800 with 73 per cent polymorphonuclears. The guaiac test on the stool was negative. A 1:20,000 tuberculin test was negative, and the sedimentation rate was 0.4 mm. per minute. A blood Hinton test was negative.

X-ray films of the chest showed a homogeneous mass occupying the upper half of the left chest cavity. It had a well-defined, convex, lower border which reached a lower level medially than laterally. It could not be separated from the mediastinal shadow and touched the chest wall laterally. On the lateral view it occupied the whole anteroposterior diameter of the upper portion of the chest. In the midportion of the chest a definite, slightly lobulated, anterior border could be seen and an indefinite posterior border. There was questionable calcification in the lateral posterior aspect of the mass. The left lower lung field was slightly brighter than the right, and the left diaphragm was in normal position. The trachea was displaced slightly to the right and anteriorly, without definite pressure defect. The left main bronchus was displaced slightly downward without evidence of obstruction of it or of the beginning of the upper or lower lobe bronchi. The dorsal spine showed no evidence of disease except for slight scoliosis to the left in the upper dorsal region. The ribs were slightly more separated than normal but not eroded.

On the tenth day an operation was performed.

Differential Diagnosis

Dr. John D. Stewart: In summary, we have as a diagnostic problem a patient who has no symptoms of disease but who was discovered during the course of routine studies to have a mass filling the upper half of the left hemithorax. Incidentally, the campaign against tuberculosis in schoolchildren, through which chest x-ray studies are made in case a routine tuberculin test is positive, has led to the discovery of a good deal of nontuberculous disease.

What lesions could cause this picture? In general terms such a mass is inflammatory, cystic or neoplastic, and lies either inside or outside the lung. The evidence in this case is altogether against inflammation. There is no impairment of general health, no loss of weight or strength. There is absence of fever, anemia and leukocytosis, and the sedimentation rate is normal. The large size of the mass and its smooth outline by x-ray also speak against an inflammatory origin. For the same reason cold abscess seems unlikely, and the negative 1:20,000 tuberculin test supports this conclusion. One wonders how the positive tuberculin test which resulted in the discovery of the intrathoracic mass was done. To be sure the mass in this case is in intimate relation with the mediastinum, and tuberculous mediastinal lymph nodes do sometimes caseate and form a mediastinal shadow in the x-ray. However, this mass fills the entire upper half of the left hemithorax, it is too large and too eccentric, and there is no involvement of the chest wall.

Could the lesion be a cyst, such as a congenital cyst, an echinococcus cyst, a bronchiogenic cyst or a dermoid cyst? All such cysts in the absence of secondary infection or communication with a bronchus may reach a large size without producing symptoms. The suggestion of calcification in the lateral posterior aspect of the mass is consistent with the diagnosis of a cyst with a partially calcified wall. The extent of the shadow suggesting calcification is not stated, but the possibility of a tooth in a dermoid cyst comes to mind. However, the x-ray appearance of the mass, to my mind, is against the diagnosis of cyst. The outline of the mass is not spherical but runs downward and medially almost in a straight line. Dermoid cyst, perhaps the commonest cyst arising in the mediastinum, as a rule takes origin in the anterior mediastinum. In this case the mass fills the entire

upper thorax in the lateral x-ray view, and position gives no lead in this connection.

In my opinion the most likely diagnosis is a benign, solid tumor of mediastinal origin. In favor of this diagnosis are the good general state of health of the patient, absence of bronchial, pulmonary, or esophageal symptoms, absence of evidence of invasions of nerves, blood vessels or lymphatics and negative laboratory studies. In favor also are the x-ray appearance of homogeneity and smooth outline, the solitary nature of the tumor and the absence of pleural fluid and of rib involvement.

In considering benign mediastinal tumors of the upper and even the lower thorax, substernal or "plunging" goiter has to be kept in mind. No mention is made of the thyroid gland in the physical examination, but presumably it felt normal. An intrathoracic goiter may sometimes appear to be detached from the thyroid gland proper, but as a rule the connection can be made out on palpation deep in the neck. The large size of the mass, its asymmetrical situation and its position in the posterior as well as the anterior upper thorax, all are against the diagnosis of intrathoracic goiter.

In view of the x-ray findings in which no changes in the thoracic cage are demonstrated, tumor such as chondroma or chondrosarcoma, arising in the thoracic bones, can be disregarded. Absence of defect or displacement in the thoracic spine and ribs, as demonstrated by x-ray, is evidence against the so-called "hour-glass" tumor described by Heuer,* consisting of a neurofibroma or lipomatous mass partly inside the thorax and encroaching on the vertebral column.

Perhaps the commonest mediastinal tumor is a tumor of neurogenic origin, such as neurofibroma or ganglioneuroma, and this seems to me the best diagnosis in this case. The intrathoracic neurofibroma may reach a large size, and there may or or may not be paralysis or pain from the nerve of origin. Frequently, however, the neurofibromas are multiple, and can be found outside the thorax as well. In this particular case ganglioneuroma is the more likely diagnosis. The absence of Horner's syndrome is against ganglioneuroma of high sympathetic origin. Such tumors are usually slowly growing, and frequently found in young people with unimpaired general health.

Clinical Discussion

Dr. Edward D. Churchill: Our preoperative diagnosis was neurofibroma or ganglioneuroma. The differential diagnosis between these two tumors is usually possible only under the microscope unless there is definite evidence of sympathetic-trunk paralysis.

Dr. Tracy B. Mallory: In how many cases have you seen that?

Dr. Churchill: Only in one. In our last case we made a definite diagnosis of ganglioneuroma.

Dr. Mallory: To distinguish between the different types of tumors which arise from nerve and nerve ganglions is after all sometimes difficult even for the histologist and is usually not a matter of great clinical importance. I confidently expect, however, that the radiologist will soon be able to give us more help in determining the origin of some of the posterior mediastinal tumors in the sympathetic cord. The tumors tend to grow along the rami to the spinal nerve root and in so doing must markedly enlarge the vertebral foramens. It seems as though the secondary bone deformity should be recognizable. Would you agree, Dr. Churchill?

Dr. Churchill: Yes, I think, however, that you ought to make it clear that this is not invasion of the bone but purely a pressure deformity.

A Physician: What are the probabilities of the tumor's extending along the nerve root into the spinal canal to a point where you cannot reach it?

Dr. Churchill: The neurofibromas do that fairly frequently. The ganglioneuromas are less apt to. Sometimes, of course, tumors of the hourglass or dumbbell shape develop with both intraspinal and extraspinal tumor tissue. In such cases there may be enormous dilatation of the foramen which may become as large as a ten-cent piece.

This patient was operated on through a posterolateral incision. The fifth rib was excised, and its posterior end was found to be flattened in a horizontal plane. This made the diagnosis of a tumor of neurogenic origin very likely. The chest was opened and the fourth, sixth and seventh ribs cut posteriorly in order to give additional exposure. A large tumor mass the size of a grapefruit was found. It was completely retropleural. All the ribs overlying it were deformed into thin keels imbedded in sulci of the tumor. It appeared to arise from the fourth, fifth and sixth intercostal nerves. At one point it extended into a vertebral foramen which was at least 1.5 cm. in diameter. When the tumor was dissected out a pulsating structure, probably representing dura, was visible.

Preoperative Diagnosis

Mediastinal tumor: neurofibroma? ganglioneuroma?

*Heuer, G. J.: Thoracic lipomas. Ann. Surg. 98:801-819, 1933.

Dr. Stewart's Diagnosis

Benign mediastinal tumor of neurogenic origin, probably ganglioneuroma.

Anatomical Diagnosis

Ganglioneuroma.

Pathological Discussion

Dr. Mallory: This large tumor consisted histologically of a complicated mixture of whorls of fibrous tissue, well-formed nerve bundles and, in certain places, masses of ganglion cells. It is worth remembering from the histological point of view that the ganglion cells in such a tumor are most irregularly distributed and that it may be necessary to cut several sections before a focus of them is found.

The relation of these tumors to the so-called neuroblastomas is of considerable interest. Both may be regarded as true tumors of nerve cells: the ganglioneuroma representing the differentiated and therefore benign form, the neuroblastoma the undifferentiated and therefore malignant form. Variations in the degree of differentiation are not ordinarily observed, but the relation between the two tumors is shown by the cases in which combined areas of neuroblastoma and ganglioneuroma occur in the same tumor.

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PHYSICIANS AND THE COMPULSORY LICENSING OF NURSES

The bills on nursing which are the subject of discussion at the public hearings now being held throughout the State by an Interim Commission of the Legislature recommend a change in policy on the part of the Commonwealth. At the present time almost anyone may nurse in Massachusetts if the patient is willing and if the physician who is in charge of the patient gives his consent.

The bills contain the prohibition that, with a few specific exceptions, no one unless licensed by the State shall nurse the sick. The conditions of licensure include certain qualifications on the part of the candidate for the practice of nursing.

The purpose of such compulsory licensing is of course the protection of the public, and, reasoning by analogy, it may be said that such protection is just as important in nursing as it is in medicine. But the analogy is not perfect, for the initiative for treatment rests with the physician and the nurse practically always works under his supervision and direction. If she does not render nursing service in a satisfactory manner it is his duty to see that she is replaced by someone who will give adequate care.

It is at this point that a responsibility of the medical profession should be pointed out. If physicians think a higher standard of nursing is required than now obtains and that, in the less skilled types of nursing service, standards of qualification should be set by the State, with exclusion of the incompetent, they should make their opinions known to the Commission. If they know of any patients who have suffered because of incompetent nursing they should inform the Commission of this fact.

A fundamental requirement of an adequate bill which prohibits the practice of nursing except by licensed persons is a definition of what is included in the prohibition, that is to say, a definition of nursing. Neither bill meets this requirement and both are very weak when they attempt to distinguish between a nurse and an attendant on the basis of what each may do. This is the critical point of any bill. It may be decided that it is wise to license nurses at any number of levels of nursing service, — the bills under consideration name two, — but a definition of nursing is always required.

The composition of the board is another point in the bill of interest to physicians. Of a proposed board of seven members only one is to be a physician — the secretary of the Board of Registration in Medicine. At the present time in a five-member board two are physicians — the secretary, as above, and the superintendent of a hospital having a school of nursing.

The circumstances under which the Board of Registration of Nurses was created thirty years ago are not clear, but medical education then, though subject to some adverse criticism, had not yet felt the forcible impact of the Carnegie Report, and certainly the training of the nurse as an educational procedure was looked on seriously by few

persons. However, the necessary interest of the medical profession in nursing was recognized by the creation of a board in which two fifths of the members were physicians.

In the past thirty years medical education has made tremendous advances. No just observer of nursing education would claim corresponding progress for nursing, in spite of what has actually been accomplished. Perhaps lack of contact between leaders in the two groups has been the responsible factor. In any case, the great interest of the physician in the nursing care that his patient receives, since he has the prime responsibility for the patient's welfare, makes necessary a careful consideration of how much formal responsibility he should have in the training and education of the nurse, in determining her fitness for practice, and in ascertaining her qualifications for license. The provisions of the bills which have been drafted seem to undervalue the part which he ought to take.

Although the bills which are under consideration have been drafted by the Attorney General and by the State Nurses' Association, the more interested persons are the patients — the sick public — and the physicians who have the chief responsibility for their care. It is possible that if the standards for admission to the practice of medicine in Massachusetts were raised to a reasonable level, instead of being the lowest in the United States, the standards of nursing practice would be automatically raised at the insistence of better doctors, without change in the statute. Are physicians really interested in nursing?

KNITTING THE RAVELED SLEEVE

An article by Lois Mattox Miller, in the July issue of *Hygeia,* warns against the increased use of the barbiturates as self-administered hypnotics. Sleeping pills, it is said, are now as good sellers as aspirin and laxatives, and contrary to general belief, they are habit forming. For every patient who takes a hypnotic under a physician's direction, there are dozens in every walk of life who buy their sleep medicine over the counter on their own responsibility.

Our present era, demarcated from the previous one by the World War, using that episode in international relations as a convenient milestone, might be called the era of human tension, or the jittery age. Before the war, life contrived to be comfortable — if a bit slow by modern standards — for many people. The war itself was a period of tenseness, of excitement, of patriotic fervor. During it the cigarette really came into its own.

Life can hardly be said to have been socially or economically "comfortable" since that time. Men, and women, have had much to meet in the way of hopes and fears and disappointments. Apprehension, insecurity and worry have been their frequent bedfellows, and they do not make good sleeping companions.

The cigarette is a sort of ground wire through which excessive nervous energy is discharged; alcohol provides an artificial relaxation and an escape from reality; and at the end of the day the hypnotic drug provides final surcease of all that is disagreeable — a sleep and a forgetting. Unfortunately the use of all of these props to living is on the increase.

The warning is a timely one, even if a little overdramatized. Samples of drugs that put us to sleep and drugs that keep us awake are flooding our professional desks. In lieu, for the present, of adequate legislation to control the sale of these medicines, our profession must accept a degree of responsibility for the manner in which they are prescribed and reiterate a warning against their indiscriminate use.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

TRAUMATIC RUPTURE OF THE UTERUS FOLLOWING VERSION

Mrs. M. T., a thirty-four-year-old gravida VII, was admitted to the hospital on August 23, 1930, for an elective induction.

The family history was essentially negative. The patient's past history was also negative. Catamenia began at thirteen, were regular, with a twenty-eight-day cycle, and three to four days in duration, and were unaccompanied by pain. Her last period began November 18, 1929, making her expected date of confinement August 28. Two previous pregnancies had resulted in miscarriages for which she had been curetted. She had had two versions, a difficult forceps and a breech extraction for her remaining four pregnancies.

When she was first seen on April 11, a physical examination was perfectly normal except for a very badly torn and scarred cervix. Her prenatal course was normal and uneventful. On entrance to the hospital she was given castor oil and quinine. Under nitrous-oxide and oxygen anesthesia the membranes were ruptured at 10:15 a. m. At this time she was in definite labor. The vertex was presenting; the cervix was dilated three fingers and was very irregular because of an extensive tear on the left which ran up to the vaginal vault. She was fully dilated about 12:30 p. m., and because of the posterior position and a moderately flat pelvis, it was decided to deliver her by version. The turning of the baby was not difficult; the aftercoming head, however, could not be delivered by suprapubic pressure so a Piper forceps was finally employed. The baby, a girl, weighed 8 lb. 6 oz.

Immediately after delivery a large hematoma was noticed on the left vulva. The placenta did not come away and was removed manually an hour later. At that time a large tear was discovered in the left vaginal vault. A diagnosis of traumatic rupture of the lower uterine segment, due to a cervical scar, was made. Following the removal of the placenta, the patient became cold and pulseless and perspired profusely. She was given two transfusions of 500 cc. of citrated blood each, and laparotomy was immediately undertaken. The abdomen was opened in the midline, and a large amount of blood and blood clots were found on both sides of the uterus but more particularly on the left. On examination there was a large rent on the left and posterior aspect of the lower segment of the uterus, involving the left broad ligament and the posterior wall of the vagina. A supravaginal hysterectomy was quickly done, and a gauze drain was left in the vagina.

For five days she had a very smooth convalescence. At the end of that time she developed a temperature associated with diarrhea, which spontaneously cleared up. The patient was discharged on the twenty-second postoperative day, with a well baby.

Comment. Cervical scars may predispose to rupture of the uterus. The difficulty of getting the head through the cervix undoubtedly caused this particular rupture. The appearance of the large hematoma in the vulva on the left was a very suggestive sign of rupture. When the diagnosis of rupture was made, laparotomy was the operation of choice. The tear could not be sutured vaginally. The extent of the rupture revealed at laparotomy made hysterectomy the only adequate procedure.

A series of selected case histories by members of the section will be published weekly.

Comments and questions by subscribers are solicited and will be discussed by members of the section.

APPLICANTS FOR FELLOWSHIP

PUBLISHED IN ACCORDANCE WITH THE PROVISIONS OF THE BY-LAWS (CHAPTER I, SECTION 1) AS AMENDED JUNE 2, 1938

BARNSTABLE DISTRICT

BULLOCK, DONALD S., 15 Main Street, Falmouth.
Tufts College Medical School, 1937.

RICE, JACKSON M., 464 Main Street, Hyannis.
Middlesex College of Medicine and Surgery, 1932.

Donald E. Higgins, *Secretary.*

BERKSHIRE DISTRICT

BRESLIN, JAMES E., 285 Pomeroy Avenue, Pittsfield.
Johns Hopkins University School of Medicine, 1925.

CARROLL, FRANCIS B., 226 Stockbridge Road, Great Barrington.
University of Rochester School of Medicine, 1935.

CHESANOW, ALBERT, 133 Castle Street, Great Barrington.
Middlesex College of Medicine and Surgery, 1928.

FIERMAN, JOSEPH H., Becket.
Boston University School of Medicine, 1935.

GOODMAN, HARRY, 28 Bank Row, Pittsfield.
Tufts College Medical School, 1931.

GOSPODAREK, FRANCIS S., 56 Summer Street, Adams.
College of Physicians and Surgeons, Boston, 1926.

GREENGOLD, DAVID B., St. Luke's Hospital, Pittsfield.
University of Maryland School of Medicine and the College of Physicians and Surgeons, 1936.

MASSIMIANO, ANTONIO, 100 North Street, Pittsfield.
Columbia University, College of Physicians and Surgeons, 1935.

Hugh J. Downey, *Secretary.*

BRISTOL NORTH DISTRICT

ELIAS, ANTHONY N., 98 East Water Street, Taunton.
Georgetown University School of Medicine, 1937.
FABER, DAVID, 7 Creedway, Taunton.
Tufts College Medical School, 1935.

William H. Swift, *Secretary*.

BRISTOL SOUTH DISTRICT

BARRETT, JAMES A., 7 Middle Street, South Dartmouth.
Maryland Medical College, 1910.
DE MELLO, MANUEL F., 198 County Street, New Bedford.
Georgetown University School of Medicine, 1937.
DUEST, LLOYD J., 171 Elm Street, New Bedford.
Tufts College Medical School, 1937.

Albert H. Sterns, *Secretary*.

ESSEX NORTH DISTRICT

BAIN, DAVID J., Lawrence General Hospital, Lawrence.
Tufts College Medical School, 1938.
BARONE, SALVATORE A., 169 Haverhill Street, Lawrence.
Middlesex College of Medicine and Surgery, 1930.
BARRY, JOHN M., Lawrence General Hospital, Lawrence.
Georgetown University School of Medicine, 1938.
DERBY, EDWARD A., 9 Campo Seco Street, Lawrence.
Georgetown University School of Medicine, 1936.
EWELL, JOHN W., The Close, Rowley.
Harvard Medical School, 1936.
FEINMAN, MAXWELL H., 97 Newton Street, Lawrence.
Boston University School of Medicine, 1936.
HUMPHREYS, STORER P., 155 High Road, Newbury.
Yale University School of Medicine, 1932.
MEISTER, ABRAHAM J., 335 Howard Street, Lawrence.
University of Vermont College of Medicine, 1936.
WHITTEN, JAMES F., 39 Market Street, Amesbury.
Harvard Medical School, 1936.
ZANNINI, NICHOLAS, 203 Haverhill Street, Lawrence.
Middlesex College of Medicine and Surgery, 1929.

Elmer S. Bagnall, *Secretary*.

ESSEX SOUTH DISTRICT

BALKUS, VINCENT A., 21 Summer Street, Danvers.
Tufts College Medical School, 1936.
BERNHARDT, HENRY M., 51 Humphrey Street, Marblehead.
Boston University School of Medicine, 1935.
BUONICONTO, PASQUALE, 74 Elm Street, Danvers.
Royal University of Naples, Faculty of Medicine, 1935.
CHAMBERLAIN, CALVIN B., 67 Pleasant Street, Marblehead.
Tufts College Medical School, 1936.
COLTON, HUBERT P., Essex Sanatorium, Middleton.
Tufts College Medical School, 1918.
CURLEY, CORNELIUS C., 73 Lawton Avenue, Lynn.
Georgetown University School of Medicine, 1937.
DRETLER, WILLIAM M., 129 Pleasant Street, Marblehead.
Tufts College Medical School, 1935.
GARDNER, ABRAHAM, Danvers State Hospital, Danvers.
University of Vermont College of Medicine, 1936.
GREEN, HARRY, 31 Middle Street, Gloucester.
Massachusetts College of Osteopathy, 1929.
Middlesex College of Medicine and Surgery, 1932.
LARCHEZ, HENRY F., Walnut Road, South Hamilton.
Middlesex College of Medicine and Surgery, 1932.
SESEN, HAROLD M., 59 Baltimore Street, Lynn.
Tufts College Medical School, 1931.
WILSON, STANLEY A., Salem Hospital, Salem.
University of Vermont College of Medicine, 1920.

John R. Shaughnessy, *Secretary*.

FRANKLIN DISTRICT

FOOTNICK, SAMUEL, 30 East Main Street, Orange.
Middlesex College of Medicine and Surgery, 1930.
GOULD, GEORGE K., Millers Falls.
Middlesex College of Medicine and Surgery, 1933.

Charles Moline, *Secretary*.

HAMPDEN DISTRICT

ABBOT, EDWARD A., 225 High Street, Holyoke.
Boston University School of Medicine, 1935.
CARSON, PARKER C., Shriners' Hospital, Springfield.
Baylor University College of Medicine, 1935.
CLEVELAND, HAROLD F., 462 Belmont Avenue, Springfield.
Middlesex College of Medicine and Surgery, 1920.
CONWAY, WILLIAM S., 646 Carew Street, Springfield.
University of Maryland School of Medicine and the College of Physicians and Surgeons, 1910.
DRISCOLL, JOHN J., 229 Chestnut Street, Holyoke.
Tufts College Medical School, 1937.
FANELLI, RAYMOND J., 62 Main Street, West Springfield.
Georgetown University School of Medicine, 1935.
FEDERICI, LOUIS M. N., 76 Woodside Terrace, Springfield.
College of Physicians and Surgeons, Boston, 1923.
FIENBERG, ROBERT, Westfield State Sanatorium, Westfield.
Harvard Medical School 1932.
FORTIN, FRANCIS F., 6 Carlisle Street, Springfield.
Jefferson Medical College of Philadelphia, 1932.
HUSTON, LEWIS L., 267 Longhill Street, Springfield.
Harvard Medical School, 1934.
KOTARSKI, WILLIAM C., 700 Chestnut Street, Springfield.
Middlesex College of Medicine and Surgery, 1929.
MERICAN, MILTON, 225 Chestnut Street, Holyoke.
St. Louis College of Physicians and Surgeons, 1924.
PENDOLA, ANTHONY S., 139 Chestnut Street, Holyoke.
Middlesex College of Medicine and Surgery, 1920.
RUSSOLILLO, FRANCIS E., 12 Congress Street, Westfield.
George Washington University Medical School, 1936.
SCOLA, JAMES V., 30 Park Street, West Springfield.
New York Homeopathic Medical College and Flower Hospital, 1936.
SHERMAN, DAVID E., 447 Sumner Avenue, Springfield.
Middlesex College of Medicine and Surgery, 1930.

Hervey L. Smith, *Secretary*.

HAMPSHIRE DISTRICT

HOLDEN, EUGENE M., 86 North Pleasant Street, Amherst.
Tufts College Medical School, 1937.
MAKOFSKI, ANTHONY B., 15 King Street, Northampton.
Kansas City University of Physicians and Surgeons, 1929.
PARKER, RUTH, Northampton State Hospital, Northampton.
Woman's Medical College of Pennsylvania, 1932.

Joseph D. Collins, *Secretary*.

MIDDLESEX EAST DISTRICT

ANDREN, HENRY E., New England Sanitarium and Hospital, Stoneham.
College of Medical Evangelists, 1937.

KELEHER, PAUL C., 48 Pleasant Street, Woburn.
Harvard Medical School, 1936.

LAND, HERBERT, Bow Street, North Reading.
Middlesex College of Medicine and Surgery, 1930.

MORELLI, DARIO, 26 Richardson Avenue, Wakefield.
Royal University of Naples (Italy), 1932.

NICKERSON, DONALD A., 25 Lynn Fells Parkway, Melrose.
Tufts College Medical School, 1933.

RHOAD, OWEN W., 50 Chute Street, Reading.
Northwestern University Medical School, 1934.

Kenneth L. Maclachlan, *Secretary*.

MIDDLESEX NORTH DISTRICT

FLANAGAN, JAMES E., St. Joseph's Hospital, Lowell.
Tufts College Medical School, 1938.

HOYE, CHARLES E., St. Joseph's Hospital, Lowell.
Tufts College Medical School, 1938.

PERRAULT, GERARD R., St. Joseph's Hospital, Lowell.
Tufts College Medical School, 1938.

Theodore A. Stamas, *Secretary*.

MIDDLESEX SOUTH DISTRICT

BALDAUF, LEON K., Ambassador Hotel, Cambridge.
Johns Hopkins University School of Medicine, 1905.

BURKE, FRANCIS M., 12 West Central Street, Natick.
Harvard Medical School, 1936.

CAMMISA, JAMES J. V., 1872 Commonwealth Avenue, Allston.
Harvard Medical School, 1928.

CASTLEMAN, BENJAMIN, 46 Horne Road, Belmont.
Yale University School of Medicine, 1931.

CURRIER, GEORGE E., Sudbury.
Tufts College Medical School, 1936.

DAWSON, WILLIAM E., Cambridge Hospital, Cambridge.
Harvard Medical School, 1935.

DE FOREST, GIDEON K., Nine Acre Corner, Concord.
Yale University School of Medicine, 1932.

FOSTER, SAWYER, 6 Pemberton Road, Cochituate.
Tufts College Medical School, 1936.

GAHAN, HENRY M., 19 Washington Street, Medford.
McGill University Faculty of Medicine, 1935.

HOFFMAN, PHOEBE D., 7 Park Circle, Arlington Heights.
Cornell University Medical College, 1920.

HOUGHTON, JOHN D., 152 Suffolk Road, Chestnut Hill.
Harvard Medical School, 1932.

KING, FRANCIS B., 2 Newport Road, Cambridge.
Tufts College Medical School, 1934.

KRONBERGER, ALFRED M., 7 Park Avenue, Newton.
College of Physicians and Surgeons, Boston, 1932.

LEVIN, HARRY M., 109 Beach Street, Malden.
College of Physicians and Surgeons, Boston, 1933.

MCGIRR, JOHN C., 1436 Cambridge Street, Cambridge.
Harvard Medical School, 1936.

MONAHAN, DAVID T., Forrester Road, Belmont.
Yale University School of Medicine, 1933.

NANCE, WILLIAM K., 7 Park Circle, Arlington.
Vanderbilt University School of Medicine, 1924.

RIEMER-RUBENSTEIN, DELILAH, 228 Foster Street, Brighton.
Woman's Medical College of Pennsylvania, 1936.

ROBINSON, HOWARD E., 12 Cottage Street, Cambridge.
Boston University School of Medicine, 1937.

ROSE, AUGUSTUS S., 17 Cross Street, Belmont.
Harvard Medical School, 1932.

SAIPE, JULIUS, 240 Highland Avenue, Somerville.
Middlesex College of Medicine and Surgery, 1933.

STOLLER, HARRY I., 406 Ferry Street, Everett.
Middlesex College of Medicine and Surgery, 1930.

THOMAS, JOHN D., 10 Ellery Street, Cambridge.
College of Physicians and Surgeons, Boston, 1929.
University of Lausanne, 1935.

WIKLUND, FOLKE W., 332 Pleasant Street, Malden.
Tufts College Medical School, 1935.

Alexander A. Levi, *Secretary*.

NORFOLK DISTRICT

BEASER, SAMUEL B., 14 Browne Street, Brookline.
Columbia University, College of Physicians and Surgeons, 1934.

BENNETT, HENRY S., 25 Shattuck Street, Roxbury.
Harvard Medical School, 1936.

CAROLAN, JAMES F., State Prison Colony, Norfolk.
Tufts College Medical School, 1935.

CROSKERY, WILLIAM F., 261 Reedsdale Road, Milton.
Boston University School of Medicine, 1937.

DI RAGO, JOSEPH V., 44 York Terrace, Brookline.
Middlesex College of Medicine and Surgery, 1933.

FAILLACE, FEDELE M., 44 Penniman Road, Brookline.
Royal University of Rome, Faculty of Medicine and Surgery (Italy), 1934.

FAILLACE, GAETANO, 44 Penniman Road, Brookline.
Royal University of Naples, Faculty of Medicine and Surgery (Italy), 1902.

HARRIS, HERBERT I., 38 Webster Place, Brookline.
Tufts College Medical School, 1936.

HELWIG, ELSON B., 179 Kent Street, Brookline.
Indiana University School of Medicine, 1932.

LAPIDUS, BERNARD, 110 Howland Street, Roxbury.
Tufts College Medical School, 1937.

LONG, ARTHUR P., 198 Fair Oaks Park, Needham.
State University of Iowa College of Medicine, 1934.

MALTBY, GEORGE L., 30 Jamaicaway, Jamaica Plain.
Harvard Medical School, 1936.

MCKEE, DAVID O., 8 Lorette Street, West Roxbury.
Tufts College Medical School, 1932.

MEDINGER, FREDERICK G., 274 Brookline Avenue, Roxbury.
University of Pennsylvania School of Medicine, 1934.

MORRISON, HARVEY R., 106 Sumner Road, Brookline.
Harvard Medical School, 1934.

NEIMANT, JOHN A., 180 Norfolk Street, Dorchester.
Tufts College Medical School, 1936.

PHILLIBERT, CUTHBERT E. C., 221 Walnut Avenue, Roxbury.
Tufts College Medical School, 1931.

RUBIN, ISRAEL, 400 Walk Hill Street, Dorchester.
University of Leipzig, 1933.

SIMPSON, MARGARET R., Boston State Hospital, Dorchester Center.
Boston University School of Medicine, 1934.

SMALL, GEORGE A., 106 Walpole Street, Norwood.
Tufts College Medical School, 1936.

TALBOT, NATHAN B., 10 Cottage Farm Road, Brookline.
Harvard Medical School, 1936.

TALKOV, ROBERT H., 57 Lithgow Street, Dorchester.
Tufts College Medical School, 1937.

VOGEL, ERNEST J., 1690 Centre Street, West Roxbury.
Harvard Medical School, 1936.

WARNSHUIS, FREDERICK C., 24 Dean Road, Brookline.
Grand Rapids Medical College, 1902.

WEXLER, JACOB, 967 Blue Hill Avenue, Dorchester.
Middlesex College of Medicine and Surgery, 1933.

WHELAN, CHARLES S., Boston Lying-in Hospital, Roxbury.
Harvard Medical School, 1933.

WYATT, GEORGE M., 370 Longwood Avenue, Roxbury.
Western Reserve University School of Medicine, 1933.

Frank S. Cruickshank, *Secretary*.

NORFOLK SOUTH DISTRICT

CAMERON, JAMES T., 67 Union Street, South Weymouth.
Tufts College Medical School, 1936.

FRANKMAN, WILLIAM, 106 Billings Road, Quincy.
St. Louis College of Physicians and Surgeons, 1921.

GURWITZ, JACK, Quincy City Hospital, Quincy.
Tufts College Medical School, 1938.

HELFRICH, WILLIAM R., 206 Manet Avenue, Quincy.
Boston University School of Medicine, 1936.

SMITH, JOHN E., 285 Front Street, Weymouth.
Tufts College Medical School, 1935.

THOMPSON, ALFRED P., 50 Brook Street, Wollaston.
Boston University School of Medicine, 1897.

VILLONE, ANTHONY J., 65 School Street, Quincy.
Tufts College Medical School, 1918.

Robert L. Cook, *Secretary*.

PLYMOUTH DISTRICT

MURPHY, WILLIAM F., 122 Front Street, Scituate Harbor.
Tufts College Medical School, 1935.

POLLEN, DAVID A., 153 North Main Street, Middleboro.
Middlesex College of Medicine and Surgery, 1931.

STALK, THEODORE, 62 Curtis Street, Egypt.
Boston University School of Medicine, 1937.

Howard C. Reed, *Secretary*.

SUFFOLK DISTRICT

BROWN, ETHAN A., 75 Bay State Road, Boston.
St. Mary's Hospital Medical School, London, 1935.

DAWES, DANIEL C., 135 Marlboro Street, Boston.
Yale University School of Medicine, 1929.

DAWES, LYDIA M. G., 135 Marlboro Street, Boston.
Yale University School of Medicine, 1929.

DUSHANE, JOSEPH E., Boston City Hospital, Boston.
Tufts College Medical School, 1936.

HOMMEL, EDWARD H., 186 Dorchester Street, South Boston.
Middlesex College of Medicine and Surgery, 1931.

KELLEY, CHARLES H., 36 Hancock Street, Boston.
Harvard Medical School, 1934.

NORCROSS, NATHAN C., 3400 Spruce Street, Philadelphia, Pa.
Boston University School of Medicine, 1932.

PETERS, CAREY M., 2 Autumn Street, Boston.
Harvard Medical School, 1936.

SHUMAN, HAROLD I., Boston City Hospital, Boston.
Boston University School of Medicine, 1935.

SHUMAN, HYMAN H., Boston City Hospital, Boston.
University of Vermont Medical College, 1935.

SKLAVER, JOSEPH, Boston City Hospital, Boston.
University of Michigan Medical School, 1937.

SMITH, PATRICIA H., 7 Grove Street, Boston.
Boston University School of Medicine, 1936.

VILKER, ARTHUR H., 1510 North Shore Road, Revere.
Middlesex College of Medicine and Surgery, 1930.

WEINSTEIN, BENJAMIN L., 70 Campbell Avenue, Revere.
Tufts College Medical School, 1935.

ZOLLO, FELICE J., 353 Revere Street, Revere.
Middlesex College of Medicine and Surgery, 1928.

John P. Monks, *Secretary*.

WORCESTER DISTRICT

COHEN, LOUIS H., Worcester State Hospital, Worcester.
Yale University School of Medicine, 1931.

CROCE, EDMUND J., 36 Shelby Street, Worcester.
Harvard Medical School, 1935.

FEINSILVER, OSCAR, Rutland State Sanatorium, Rutland.
Tufts College Medical School, 1935.

GORMAN, RICHARD J., Worcester City Hospital, Worcester.
Tufts College Medical School, 1935.

HAGBERG, WALDO O., 29 Ekman Street, Worcester.
Massachusetts College of Osteopathy, 1929.
Middlesex College of Medicine and Surgery, 1932.

HAGERTY, EDWARD D., Worcester City Hospital, Worcester.
McGill University Faculty of Medicine, 1936.

HENDERSON, JOHN W., Jr., Worcester City Hospital, Worcester.
Harvard Medical School, 1935.

KINIRY, B. JOHN, Worcester City Hospital, Worcester.
Tufts College Medical School, 1936.

LEBEAUX, LINCOLN, Shrewsbury.
University of Michigan Medical School, 1937.

LENTINO, JOSEPH W., 85 Walnut Street, Clinton.
Royal University of Rome Medical School, 1934.

MULVIHILL, WALTER M., 981 Main Street, Worcester.
Boston University School of Medicine, 1936.

NUGENT, RICHARD S., Worcester City Hospital, Worcester.
Tufts College Medical School, 1936.

WILLARD, RAYMOND D., Jr., 1006 Main Street, Worcester.
Tufts College Medical School, 1936.

George C. Tully, *Secretary*.

WORCESTER NORTH DISTRICT

AREY, JEANNIE M., 107 Edgell Street, Gardner.
Tufts College Medical School, 1914.

HITCHCOCK, JAMES, 67 Prichard Street, Fitchburg.
Harvard Medical School, 1922.

SILVER, JOSEPH M., 46 Prichard Street, Fitchburg.
Middlesex College of Medicine and Surgery, 1933.

WASSER, LOUIS, 5 Memorial Street, Baldwinsville.
Middlesex College of Medicine and Surgery, 1933.

Edward A. Adams, *Secretary Pro Tempore*.

MEDICAL POSTGRADUATE EXTENSION COURSES

Medical Postgraduate Extension Courses, according to the programs listed below, will be given this fall. The courses in the remainder of the State will be held during the winter and spring months. Announcement concerning these programs will be made in the *Journal* at a later date. Programs for the sessions this fall have been mailed to each doctor in these districts.

Bristol South District

Place: New Bedford

SUBJECT	DATE
Whooping Cough. — The Present Status of Vaccine Therapy as a Prophylactic and as a Therapeutic Measure, the Early Diagnosis by Laboratory Procedures, and the Treatment of Complications	October 28
Obstetrical Examination	November 4
Clinical Demonstrations. — One or Two Cases, Preferably Illustrating the Same Condition, to Be Selected by Local Committee and Data Given to Instructor Two Weeks Prior to Presentation	November 18
Delivery and the Puerperium	November 25
The Indications and Contraindications for Removal of Tonsils and Adenoids	December 2
Bleeding in the Third Trimester of Pregnancy	December 9
The Control and Treatment of Respiratory Infections. (This is to include the serum treatment of pneumonia in infants and children.)	December 16
The Toxemias of Pregnancy	December 23
Scurvy. — Its Early Diagnosis and Prevention	January 6
Sepsis	January 13

Meetings to be held at the St. Luke's Hospital, Fridays, at 4:00 p. m.

Howard P. Sawyer, M.D.,
Robert H. Goodwin, M.D.,
Chairmen

Note: Because of Armistice Day, the course will be omitted November 11. It will also be omitted December 30 during the Christmas holidays.

Essex North District

Place: Lawrence

SUBJECT	DATE
Bright's Disease and Hypertension. Evaluation of New Therapy: Diagnosis	October 21
Heart Disease. The Treatment of "Heart Attacks" or "Cardiovascular Emergencies"	October 28
The Control and Treatment of Respiratory Infections. (This is to include the serum treatment of pneumonia in infants and children.)	November 4
Whooping Cough. — The Present Status of Vaccine Therapy as a Prophylactic and as a Therapeutic Measure, the Early Diagnosis by Laboratory Procedures, and the Treatment of Complications	November 18
Anemia. Modern Methods in Diagnosis and Treatment of Blood Dyscrasias	November 25
Medical Complications in Pregnancy	December 2
Syphilis. Latent Syphilis. — Diagnosis and Treatment	December 9
Gonorrhea. Modern Treatment of Gonorrhea	December 16

Meetings to be held at the Lawrence General Hospital, Fridays, at 4:30 p. m.

John Parr, M.D., *Chairman*

Note: Because of Armistice Day, the course will be omitted November 11.

Essex South District

Place: Salem

SUBJECT	DATE
Anemia. Modern Methods in Diagnosis and Treatment of Blood Dyscrasias	October 25
The Toxemias of Pregnancy	November 1
Gonorrhea. Modern Treatment of Gonorrhea	November 8
Syphilis. Latent Syphilis. — Diagnosis and Treatment	November 22
The Indications and Contraindications for Removal of Tonsils and Adenoids	November 29
Bright's Disease and Hypertension. Evaluation of New Therapy: Diagnosis	December 6
Heart Disease. The Treatment of "Heart Attacks" or "Cardiovascular Emergencies"	December 13
Whooping Cough. — The Present Status of Vaccine Therapy as a Prophylactic and as a Therapeutic Measure, the Early Diagnosis by Laboratory Procedures, and the Treatment of Complications	December 20

Meetings to be held in the Nurses' Home of the Salem Hospital, Tuesdays, at 4:00 p. m.

Walter G. Phippen, M.D., *Chairman*

Note: Because of the New England Postgraduate Assembly, the course will be omitted November 15.

Hampshire District

Place: Northampton

SUBJECT	DATE
Gonorrhea. Modern Treatment of Gonorrhea	October 26
Syphilis. Latent Syphilis. — Diagnosis and Treatment	November 2
Anemia. Modern Methods in Diagnosis and Treatment of Blood Dyscrasias	November 9
Heart Disease. The Treatment of "Heart Attacks" or "Cardiovascular Emergencies"	November 23

Bright's Disease.
Evaluation of New Therapy: Diagnosis November 30
Obstetrical Examination December 7
The Toxemias of Pregnancy December 14
The Control and Treatment of Respiratory Infections. (This is to include the serum treatment of pneumonia in infants and children.) December 21

Meetings to be held in the Nurses' Home of the Cooley Dickinson Hospital, Wednesdays, at 4:15 p. m.

Warren P. Cordes, M.D., *Chairman*

Note: Because of the New England Postgraduate Assembly, the course will be omitted November 16.

NORFOLK SOUTH DISTRICT

Place: QUINCY

SUBJECT	DATE
Syphilis. Latent Syphilis. — Diagnosis and Treatment	October 24
Gonorrhea. Modern Treatment of Gonorrhea	October 31
The Toxemias of Pregnancy	November 7
Bright's Disease and Hypertension. Evaluation of New Therapy: Diagnosis	November 21
Sepsis	November 28
Heart Disease. The Treatment of "Heart Attacks" or "Cardiovascular Emergencies"	December 5
The Indications and Contraindications for Removal of Tonsils and Adenoids	December 12
Anemia. Modern Methods in Diagnosis and Treatment of Blood Dyscrasias	December 19

Meetings to be held at the Quincy City Hospital, Whitwell Street, Quincy, Mondays, at 8:30 p. m.

David L. Belding, M.D., *Chairman*

Note: Because of the New England Postgraduate Assembly, the course will be omitted November 14.

PLYMOUTH DISTRICT

Place: BROCKTON

SUBJECT	DATE
The Toxemias of Pregnancy	October 25
The Control and Treatment of Respiratory Infections. (This is to include the serum treatment of pneumonia in infants and children.)	November 1
Heart Disease. The Treatment of "Heart Attacks" or "Cardiovascular Emergencies"	November 8
Gonorrhea. Modern Treatment of Gonorrhea	November 22
Syphilis. Latent Syphilis. — Diagnosis and Treatment	November 29
Anemia. Modern Methods in Diagnosis and Treatment of Blood Dyscrasias	December 6
Bright's Disease and Hypertension. Evaluation of New Therapy: Diagnosis	December 13
Delivery and the Puerperium	December 20

Meetings to be held in the Rosa Field Nurses' Residence, Brockton Hospital (rear of hospital), Tuesdays, at 4:00 p. m.

Walter H. Pulsifer, M.D., *Chairman*

Note: Because of the New England Postgraduate Assembly, the course will be omitted November 15.

WORCESTER DISTRICT

Place: MILFORD

SUBJECT	DATE
Medical Complications in Pregnancy	October 25
Whooping Cough. — The Present Status of Vaccine Therapy as a Prophylactic and as a Therapeutic Measure, the Early Diagnosis by Laboratory Procedures, and the Treatment of Complications	November 1
Surgical Complications in Pregnancy	November 8
Clinical Demonstrations. — One or Two Cases, Preferably Illustrating the Same Condition, to Be Selected by Local Committee and Data Given to Instructor Two Weeks Prior to Presentation	November 22
Breech Delivery	November 29
The Indications and Contraindications for Removal of Tonsils and Adenoids	December 6
Operative Obstetrics	December 13
The Control and Treatment of Respiratory Infections. (This is to include the serum treatment of pneumonia in infants and children.)	December 20
Obstetrics. — (Second part of one of above titles. Subject to be announced.)	January 3
Scurvy. — Its Early Diagnosis and Prevention	January 10

Meetings to be held in the Nurses' Home of the Milford Hospital, Tuesdays, at 8:30 p. m.

Joseph Ashkins, M.D., *Chairman*

Note: Because of the New England Postgraduate Assembly, the course will be omitted November 15. It will also be omitted December 27 during the Christmas holidays.

WORCESTER NORTH DISTRICT

Place: FITCHBURG

SUBJECT	DATE
The Control and Treatment of Respiratory Infections. (This is to include the serum treatment of pneumonia in infants and children.)	October 21
Cesarean Section, Analgesia	October 28
Bright's Disease and Hypertension. Evaluation of New Therapy: Diagnosis	November 4
Bleeding in the First Trimester of Pregnancy	November 18
Syphilis. Latent Syphilis. — Diagnosis and Treatment	November 25
Gonorrhea. Modern Treatment of Gonorrhea	December 2
Heart Disease. The Treatment of "Heart Attacks" or "Cardiovascular Emergencies"	December 9

Anemia.

Modern Methods in Diagnosis and Treatment of Blood Dyscrasias ..... December 16

Meetings to be held at the Burbank Hospital, Fridays, at 4:30 p. m.

Edward A. Adams, M.D., *Chairman*

Note: Because of Armistice Day, the course will be omitted November 11.

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FACULTY

Anemia. Chairman: Dr. William P. Murphy. Instructors: Drs. Clark W. Heath, Chester S. Keefer, George R. Minot and Maurice B. Strauss.

Bright's Disease and Hypertension. Chairman: Dr. James P. O'Hare. Instructors: Drs. Laurence B. Ellis, W. Richard Ohler and Robert S. Palmer.

Heart Disease. Chairman: Dr. Paul D. White. Instructors: Drs. Edward F. Bland, Francis L. Chamberlain, Wilfrid J. Comeau, Marshall N. Fulton, R. Earle Glendy, Ashton Graybiel, Burton E. Hamilton, T. Duckett Jones, Samuel A. Levine, Benedict F. Massell, Sylvester McGinn, Joseph H. Pratt, William D. Reid, Howard B. Sprague and Oliver H. Stansfield.

Gonorrhea. Chairman: Dr. E. Granville Crabtree. Instructors: Drs. Oscar F. Cox, Jr., Roger C. Graves and George C. Prather.

Syphilis. Chairman: Dr. E. Granville Crabtree. Instructors: Drs. William P. Boardman, Rudolph Jacoby, C. Guy Lane and Francis M. Thurmon.

Obstetrics. Chairman: Dr. Robert L. DeNormandie. Instructors: Drs. De Los J. Bristol, Jr., Christopher J. Duncan, M. Fletcher Eades, A. Gordon Gauld, Thomas R. Goethals, Roy J. Heffernan, James C. Janney, M. V. Kappius, Foster S. Kellogg, Joseph W. O'Connor, John Rock, Judson A. Smith and Raymond S. Titus.

Pediatrics. Chairman: Dr. Warren R. Sisson. Instructors: Drs. James M. Baty, Allan M. Butler, Stewart H. Clifford, Louis K. Diamond, R. Cannon Eley, Carlyle G. Flake, Joseph Garland, Harold L. Higgins, Charles I. Johnson, Charles F. McKhann, Edwin H. Place, Clement A. Smith and Edwin T. Wyman.

DEATHS

COUILLARD — EDWARD J. COUILLARD, M.D., of Main Street, Northbridge, died October 8. He was in his fifty-eighth year.

Dr. Couillard was born in Manchaug. After graduating from the Northbridge High School he attended Philips-Exeter Academy, completed two years at Dartmouth College and received his degree from the Baltimore Medical College in 1909.

Dr. Couillard held memberships in the Massachusetts Medical Society and the American Medical Association.

His widow, his mother, three sons, three brothers and four sisters survive him.

MOSES — ALVIN R. MOSES, M.D., of Charlton, died October 13. He was in his forty-third year.

Born in Lynn he was graduated from Tufts College Medical School in 1920. For eleven years he was associate medical examiner for Worcester County.

His memberships included the Massachusetts Medical Society, the American Medical Association and the New England Obstetrical and Gynecological Society.

His widow survives him.

SIMPSON — CHARLES SIMPSON, M.D., of 186 Hamilton Street, Southbridge, died October 12. He was in his sixty-third year.

Born in Southbridge he graduated from Southbridge High School and McGill University and received his degree from the University of Montreal Faculty of Medicine in 1904.

A practicing physician for thirty years, he was also a member of the Southbridge Board of Health for twelve years. Dr. Simpson was a member of the Massachusetts Medical Society and the American Medical Association.

His widow survives him.

NOTICES

INTER-STATE POSTGRADUATE MEDICAL ASSOCIATION OF NORTH AMERICA

Following is an abbreviated schedule of talks, clinics and addresses to be given at the international assembly of the Inter-State Postgraduate Medical Association at Philadelphia:

MONDAY, OCTOBER 31

Morning. "Acute Coronary Occlusion," G. Harlan Wells, Philadelphia; "The Significance of Low Back Pain," Frank R. Ober, Boston; "Present Status of the Treatment of Hirschsprung's Disease," Fred Rankin, Lexington, Kentucky; "Hydronephrosis," Herman L. Kretschmer, Chicago; "The Significance of Jaundice," Henry A. Christian, Boston.

Afternoon. "Trauma of the Larynx," Chevalier Jackson, Philadelphia; "Postoperative Complications," Elliott C. Cutler, Boston; "The Treatment of Anemia," Russell L. Haden, Cleveland; "Practical Thyroid and Pituitary Therapy in Problems of Aberrant Growth and Development," E. Kost Shelton, Los Angeles; "Syndromes of Gall Bladder Disease — Surgical Management," William D. Haggard, Nashville; "The Therapeutic Value of Blood Transfusions," Cyrus C. Sturgis, Ann Arbor; "The Surgical Management of Brain Tumors," Alfred W. Adson, Rochester, Minnesota.

Evening. "Obscure Fevers as Diagnostic Problems," George Blumer, New Haven; "Treatment of Fracture Dislocations of the Cervical Vertebrae by Skeletal Traction and Fusion," William Cone and William G. Turner, Montreal; "Complications of Pregnancy," Nicholson J. Eastman, Baltimore; "Acute Pancreatitis," Eldridge L. Eliason, Philadelphia, and William H. Erb, by invitation; "The Factors Influencing Operability and Mortality in Carcinoma of the Large Bowel," Richard B. Cattell, Boston.

TUESDAY, NOVEMBER 1

Morning. "Diagnostic Significance of Pain," Frederick J. Kalteyer, Philadelphia; "Thyroid Diseases," Robert S. Dinsmore and A. Carlton Ernstene, Cleveland; "Chronic Disease of the Liver," Charles A. Elliott, Chicago; "Clinical Significance of a Lump in the Breast," Edmond M. Eberts, Montreal; "Obstruction of the Neck of the Bladder in Men," William E. Lower, Cleveland.

Afternoon. "Tic Douloureux," Howard C. Naffziger, San Francisco; "The Treatment of Dehydration and Edema," James H. Means, Boston; "The Present Status of Our Knowledge of the Suprarenal Cortical Hormone," George A. Harrop, New Brunswick, New Jersey; "Immediate and Ultimate Prognosis in Heart Disease," Paul D. White, Boston; "Diagnosis and Treatment of Bronchio-

genic Carcinoma," Arthur C. Christie, Washington, District of Columbia; "Gastroscopy as an Aid in Diagnosis," Chevalier L. Jackson, Philadelphia; "Adenomatous Thyroid With and Without Hyperthyroidism — Medical and Surgical Aspects," Charles W. Mayo and Samuel F. Haines, Rochester, Minnesota.

Evening. "Diagnosis and Treatment of Carcinoma of the Fundus of the Uterus," Floyd E. Keene, Philadelphia; "The Present Status of Our Knowledge of Anterior Poliomyelitis," John C. Gittings, Philadelphia; "The Diagnosis and Treatment of Splenomegaly," Allen O. Whipple, New York City; "Neoplasms of the Stomach: Correlation of Roentgenological and Clinical Aspects," Fred J. Hodges, and Robert M. Bartlett, Ann Arbor; "The Use of Spinal Anesthesia," Thomas H. Russell, New York City.

WEDNESDAY, NOVEMBER 2

Morning. "Diagnosis and Treatment of Spinal Cord Tumors," Walter E. Dandy, Baltimore; "Differential Diagnosis of Acute Abdominal Disease," Claude F. Dixon, Rochester, Minnesota; "Diabetes Mellitus From an Endocrinological Viewpoint," Elliott P. Joslin, Boston; "Diagnostic Dermatological Clinic," Oliver S. Ormsby, Chicago; "Treatment of Complicated Colles Fractures," William Darrach, New York City.

Afternoon. "The Indications, Contraindications, and End Results in the Surgical Treatment of Essential Hypertension," George Crile, Cleveland; "Hyperpyrexia by Physical Agents: Technic, Indications and Results," John S. Coulter, Chicago; "Clinical Significance of Hematuria," William F. Braasch, Rochester, Minnesota; "A Medical Appraisal of the Surgery of Pulmonary Tuberculosis," William S. Middleton, Madison, Wisconsin; "The Clinical Use of Sulfanilamide in Infectious Diseases," Perrin H. Long, Baltimore; "Acute Appendicitis: Management and Mortality," George P. Muller, Philadelphia; "Injuries to the Heart. Stab Wounds, and Contusions," Claude S. Beck, Cleveland; "Improvements in Methods of Abdominal Drainage," W. Wayne Babcock, Philadelphia.

THURSDAY, NOVEMBER 3

Morning. "Role of Diseases of the Sinuses to General Medicine," Robert F. Ridpath, Philadelphia; "The Significance of Enlargement of the Abdomen in Children." Irvine McQuarrie, Minneapolis; "The Diagnosis of Bone Lesions," Dean Lewis, Baltimore; "Non-Organic Disorders of the Digestive Tract," Alfred Stengel, Philadelphia; "Arthritis with Special Reference to Etiology and Treatment," Peter T. Bohan, Kansas City, Missouri.

Afternoon. "Immediate Care of Fractures," Clay R. Murray, New York City; "Hodgkin's Disease," Warfield T. Longcope, Baltimore; "Immediate Versus Delayed Surgery in the Treatment of Acute Diseases of the Gall Bladder," Charles G. Heyd, New York City; "The Management of Peritonitis," Vernon C. David, Chicago; "The Object and the Value of Preoperative and Postoperative X-ray Treatment in Carcinoma of the Breast," George E. Pfahler, Philadelphia; "Subtemporal Decompression: Indications and Surgical Technic," Eric Oldberg, Chicago; "Relation of Trauma to Inguinal Hernia: Analysis of 1000 Herniotomies," John J. Moorhead, New York City.

Evening. "Classification and Treatment of the Epilepsies," Wilder Penfield, Montreal; "The Clinical Significance of Retinal Changes in Arterial Hypertension," Walter L. Lillie, Philadelphia; "The Prognosis and Treatment of Rheumatic Heart Disease," Fred M. Smith, Iowa City; "Psychotherapy in General Medicine," Clarence B. Farrar, Toronto; "Pellagra," John H. Musser, New Orleans.

FRIDAY, NOVEMBER 4

Morning. "Carcinoma of the Larynx with Special Reference to End Results," Louis H. Clerf, Philadelphia; "Compressed Fractures of the Vertebrae," Hubley R. Owen, Philadelphia; "The Management of Gastric and Duodenal Ulcer, Jejunal Ulcer, and Gastrojejunocolic Fistula," Frank Lahey, Boston; "The Differential Diagnosis of Diseases of the Chest and Abdomen," David Riesman, Philadelphia; "Diagnosis and Treatment of Obstructive Lesions of the Colon," John F. Erdmann, New York City.

Afternoon. "Treatment of Fractures of the Neck of the Femur by Internal Fixation," M. N. Smith-Petersen, Boston; "Complications Following Surgery of the Biliary Tract," Waltman Walters, Rochester, Minnesota; "The Diagnosis and Treatment of Peripheral Nerve Injuries," Loyal Davis, Chicago; "Obstruction of the Neck of the Bladder in Women," Hugh H. Young, Baltimore; "Studies in Growth — Precocious and Malignant," Leonard G. Rowntree, Philadelphia; "Trauma of the Abdomen," Arthur R. Metz, Chicago; "Influenzal Pneumonias: Observations on Their Pathological Features and Clinical Characteristics," Robert G. Torrey, Philadelphia.

The association, through its officers and members of the Program Committee, extends a very hearty invitation to all members of the profession in good standing in their state and provincial societies to attend the assembly. The registration fee is $5.00.

DR. ELLIOTT P. JOSLIN, *President,*
Boston, Mass.,
DR. GEORGE W. CRILE, *Chairman,*
Cleveland, Ohio,
DR. WILLIAM B. PECK, *Managing-Director,*
Freeport, Illinois.

HAMPDEN DISTRICT MEDICAL SOCIETY

The regular fall meeting of the Hampden District Medical Society will be held in the rooms of the Springfield Academy of Medicine, 20 Maple Street, Springfield, on Tuesday, October 25, at 4:15 p. m.

Dr. Henry R. Viets will speak on "Acute Lymphocytic Meningitis and Other Virus Diseases of the Central Nervous System." Discussion by fellows will follow.

Censors meet for the examination of candidates in the rooms of the Academy on Thursday, November 3, at 4:00 p. m.

HERVEY L. SMITH, *Secretary.*

MASSACHUSETTS MEDICAL BENEVOLENT SOCIETY

The annual meeting of the Massachusetts Medical Benevolent Society will be held at the Boston Medical Library, on Thursday, October 27, at 5:15 p. m.

The Council will meet at the same place at 5:00 p. m. It includes the following officers: president, vice-president, treasurer, secretary and the following trustees: Drs. J. Lewis Bremer, Lloyd T. Brown, Ernest L. Hunt, Charles C. Lund, Charles G. Mixter, John P. Monks, Walter G. Phippen, Allen G. Rice and Paul D. White.

ROBERT M. GREEN, M.D., *President,*
RICHARD CHUTE, M.D., *Secretary.*

PETER BENT BRIGHAM HOSPITAL

The Thursday afternoon clinics which are conducted by Dr. Henry A. Christian and held at the Peter Bent Brigham Hospital will be resumed at 3:30 p. m. on Thursday, January 12, 1939. Notice of each clinic will appear in the *Journal* the week before the date of the clinic.

The medical profession is cordially invited to attend.

ANNUAL SCIENTIFIC SESSION OF THE ACADEMY OF PHYSICAL MEDICINE

The Academy of Physical Medicine will hold its sixteenth annual meeting and scientific session at the Willard Hotel, Washington, District of Columbia, October 24, 25 and 26.

The program is replete with reports of recent studies in the field, every branch of which is represented. The clinical papers, research reports and scientific exhibits are contributions from outstanding leaders in their respective specialties. The medical departments of the United States Army and Navy, the Veterans' Administration, the United States Public Health Service, the Department of the Interior, other medical and scientific governmental services and the Medical Society of the District of Columbia are cooperating in the program.

A public meeting on Monday evening will be addressed by Dr. William J. Mallory, president of the Medical Society of the District of Columbia; Brigadier General Frank T. Hines, chief of the United States Veterans' Administration; Dr. Herbert P. Ramsey, chairman of the Committee on Public Information, Medical Society of the District of Columbia; Dr. Rolland A. Case, of Cleveland, president of the Academy of Physical Medicine; Dr. William D. McFee, of Boston, and Dr. William S. Bainbridge, member of the Permanent Committee of the International Congress of Military Medicine and Pharmacy.

The annual banquet will be held on Tuesday evening, October 25, when officials of the medical services in Washington and their wives will be guests of honor. President Roosevelt will be represented.

All sessions are open to the medical profession. There is no fee for registration. An attractive program has been arranged for the visiting ladies.

JOHN T. BOTTOMLEY MEDICAL SOCIETY

The first clinical meeting of the John T. Bottomley Medical Society will be held at the Carney Hospital on Wednesday, October 26, from 11:00 a. m. to 4:00 p. m. Luncheon will be served at 1:00, and a formal dinner will be held at the Copley-Plaza Hotel at 6:30 p. m.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held on Tuesday, October 25, in the Peter Bent Brigham Hospital amphitheater (Shattuck Street entrance), at 8:15 p. m. Dr. Channing Frothingham will preside.

PROGRAM

Presentation of cases.

Certain Cardiorenal Circulatory Correlations. Dr. Henry A. Christian.

Medical students and physicians are cordially invited to attend.

ROBERT ZOLLINGER, M.D., *Secretary.*

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, OCTOBER 24

TUESDAY, OCTOBER 25

*9-10 a. m. Boston Dispensary. X-ray demonstration. Dr. Alice Ettinger.
9:30 a. m. Massachusetts General Hospital. Thoracic clinic.
*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.
*8:15 p. m. Harvard Medical Society. Peter Bent Brigham Hospital amphitheater (Shattuck Street entrance).

WEDNESDAY, OCTOBER 26

8 a. m. Massachusetts General Hospital. Grand rounds. Orthopedic Department.
*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.
*12 m. Clinicopathological conference. Children's Hospital amphitheater.
*8:15 p. m. New England Heart Association. Boston Medical Library.
*8:15 p. m. Suffolk District Medical Society. Boston Medical Library.

THURSDAY, OCTOBER 27

9 a. m. Massachusetts General Hospital. Surgical grand rounds.
9 a. m. Massachusetts General Hospital. Neurological staff conference.
*9-10 a. m. Boston Dispensary. Cancer of the Lung. Dr. Richard Overholt.
*10:30 a. m. Psychiatric Rounds. Boston City Hospital. Rounds will start at Room 916 on the ninth floor of the Medical Building.
12 m. Massachusetts General Hospital. Clinicopathological conference.
5:15 p. m. Massachusetts Medical Benevolent Society. Boston Medical Library.

FRIDAY, OCTOBER 28

*9-10 a. m. Boston Dispensary. Metabolism of Bone. Dr. J. C. Aub.
10 a. m. Massachusetts General Hospital. Fracture rounds.
*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

SATURDAY, OCTOBER 29

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.
*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

OCTOBER 24-26 — Academy of Physical Medicine, Scientific Session. Washington, D. C. Notice above.

OCTOBER 24 - NOVEMBER 4 — Graduate Fortnight of the New York Academy of Medicine. Page 181, issue of August 4.

OCTOBER 25 — Harvard Medical Society. Notice above.

OCTOBER 25-28 — American Public Health Association. Page 321, issue of September 1.

OCTOBER 26 — John T. Bottomley Medical Society. Notice above.

OCTOBER 26 — New England Heart Association. Page 588, issue of October 13.

OCTOBER 27 — Massachusetts Medical Benevolent Society. Page 633.

OCTOBER 31, NOVEMBER 1, 2, 3 and 4 — Inter-State Postgraduate Medical Association. Page 451, issue of September 22, and page 632.

NOVEMBER 5 — American Board of Obstetrics and Gynecology. Page 145, issue of July 28.

NOVEMBER 5, 1938; FEBRUARY 4, MAY 15 and 16, 1939 — American Board of Obstetrics and Gynecology. Page 451, issue of September 22.

NOVEMBER 10 — Pentucket Association of Physicians, 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

NOVEMBER 15-16 — New England Postgraduate Assembly. Page vii, Advertising Section, issue of September 29.

JANUARY 12, 1939 — Peter Bent Brigham Hospital. Clinic conducted by Dr. Christian. Page 633.

MARCH 27-31, 1939 — American College of Physicians. Page 36, issue of July 7.

MAY 7-15, 1939 — International Congress of Military Medicine and Pharmacy. Page 501, issue of September 29.

SEPTEMBER, 1939 — Boston Psychoanalytic Institute. Page 450, issue of September 22.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH

NOVEMBER 2 — Beverly Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. John Homans. Subject: Clinical Forms of Thrombophlebitis with Especial Reference to the Prevention of Pulmonary Embolism.

DECEMBER 7 — Salem Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speakers: Dr. Walter G. Phippen and Dr. Channing Frothingham. Subject: Economic Problems.

JANUARY 4 — Danvers State Hospital. Clinic at 5 p. m. Dinner at 7 p. m.

FEBRUARY 8 — Essex Sanatorium, Middleton. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Edward Churchill.

MARCH 1 — Lynn Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. John Rock. Subject: Endocrinology.

APRIL 5 — Addison Gilbert Hospital, Gloucester. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Ethan Allan Brown. Subject: Allergy.

MAY 10 — Annual meeting. Salem Country Club, Peabody.

HAMPDEN

OCTOBER 25 — Page 633.

NOVEMBER 3 — Censors' meeting. Rooms of the Academy.

SUFFOLK

OCTOBER 26 — Joint meeting with New England Heart Association, Boston Medical Library, 8:15 p. m. Neurogenic Influences in Experimental Coronary Artery Disease. Dr. George E. Hall, of Toronto.

NOVEMBER 3 — Censors' meeting. 4 p. m., Boston Medical Library.

NOVEMBER 30 — Program of clinical and investigative activities. Staff of the Massachusetts General Hospital, Boston Medical Library, 8:15 p. m.

JANUARY 25, 1939 — Symposium on Diabetes. Dr. Elliott P. Joslin and associates, Boston Medical Library, 8:15 p. m.

MARCH 29 — Joint meeting with New England Pediatric Society, Boston Medical Library, 8:15 p. m. Program and speakers to be announced.

APRIL 26 — Annual meeting in conjunction with Boston Medical Library, at 8:15 p. m. Election of officers. Program and speakers to be announced.